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TRANSACTIONS

OF THE

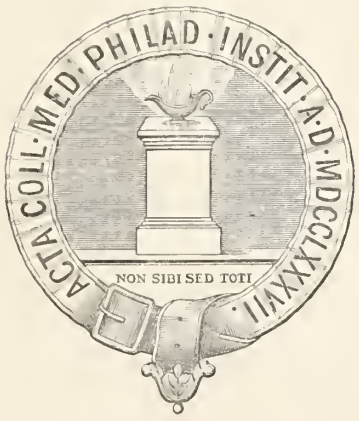
COLLEGE OF PHYSICIANS

OF

PHILADELPHIA.

THIRD SERIES.

VOLUME THE SIXTEENTH.



PHILADELPHIA:
PRINTED FOR THE COLLEGE.
1894.

COLLEGE OF PHYSICIANS OF PHILADELPHIA.

1894.

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Charles Stewart Wurts, M.D. (elected by Council March 26, 1890).

LIST

OF THE

PRESIDENTS OF THE COLLEGE FROM THE TIME OF ITS INSTITUTION.

ELECTED

1787. JOHN REDMAN.
1805. WILLIAM SHIPPEN.
1809. ADAM KUHN.
1818. THOMAS PARKE.
1835. THOMAS C. JAMES.
1835. THOMAS T. HEWSON.
1848. GEORGE B. WOOD.
1879. W. S. W. RUSCHENBERGER.
1883. ALFRED STILLÉ.
1884. SAMUEL LEWIS.
1884. J. M. Da COSTA.
1886. S. WEIR MITCHELL.
1889. D. HAYES AGNEW.
1892. S. WEIR MITCHELL.

NOTICE.

The present volume of Transactions contains the papers read before the College from January, 1894, to December, 1894, inclusive.

The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its Transactions.

Edited by
G. G. DAVIS, M.D.

DORNAN, PRINTER.

FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA.

DECEMBER, 1894.

* Non-resident Fellows.
† Fellows who have commuted dues.
° Fellows made free of dues by the College.

ELECTED

*1883. Abbot, Griffith E., Ph.D., M.D.

1892. Abbott, Alex. C., M.D., First Assistant, Laboratory of Hygiene, University of Pennsylvania.

1870. Adler, John M., M.D.

1876. Alison, Robert H., M.D.

1867. Allen, Harrison, M.D., Professor of Zoölogy and Comparative Anatomy in the University of Pennsylvania.

1873. Allis, Oscar H., M.D., Clinical Lecturer on Orthopædic Surgery in Jefferson Medical College, and Surgeon to the Hospital of the same; Surgeon to the Presbyterian Hospital.

1888. Anders, James M., M.D., Professor of Hygiene and Clinical Diseases of Children in the Medico-Chirurgical College, Philadelphia; Physician to the Philadelphia Hospital.

1869. Andrews, T. Hollingsworth, M.D., Consulting Surgeon to the Hospital of the Good Shepherd, Radnor; Medical Director of the Bureaus of Police and Fire of the Department of Public Safety.

ELECTED

*1882. Ashbridge, Richard, M.D.

1863. Ashhurst, John, Jr., M.D., Professor of Surgery in the University of Pennsylvania; Surgeon to the Pennsylvania and the Children's Hospitals; Consulting Surgeon to St. Christopher's and the Woman's Hospitals, and to the Hospital of the Good Shepherd, Radnor.

1865. Ashhurst, Samuel, M.D., Surgeon to the Children's Hospital.

1893. Ashton, Thomas G., M.D., Chief of Out-patient Medical Department, Jefferson Medical College Hospital; Demonstrator of Clinical Medicine, Jefferson Medical College, and Visiting Physician to St. Mary's Hospital, Phila.

1857. Atlee, Walter Franklin, A.M., M.D., Corresponding Member of La Société des Sciences Médicales de Lyons; Consulting Surgeon to St. Luke's Hospital, Bethlehem; Visiting Physician to the Preston Retreat.

*1852. Bache, Thomas Hewson, M.D., Rome, Italy.

1883. Baer, Benjamin F., M.D., Professor of Gynecology in the Philadelphia Polyclinic.

†1892. Baker, George Fales, B.S., M.D., Surgeon to Out-patient Department of St. Joseph's Hospital.

1879. Baker, Washington H., M.D., Obstetrician to the Maternity Hospital.

1889. Baldy, John Montgomery, M.D., Professor of Gynecology in the Philadelphia Polyclinic; Surgeon to Gynecean Hospital; Gynecologist to Pennsylvania Hospital.

1880. Bartholow, Roberts, M.D., Professor (Emeritus) of Materia Medica, General Therapeutics, and Hygiene in Jefferson Medical College.

1894. Barton, James M., M.D., Surgeon to the Jefferson Medical College Hospital and to the Philadelphia Hospital.

1883. Baum, Charles, M.D., A.M., Ph.D.

1873. Baxter, H. F., M.D.

1883. Beates, Henry, M.D.

ELECTED

1860. Benner, Henry D., M.D.

1874. Bennett, W. H., M.D., Physician to St. Christopher's Hospital for Children and to Children's Seashore House, Atlantic City.

†1884. Biddle, Alexander W., M.D.

1884. Biddle, Thomas, M.D.

*1866. Black, J. J., M.D., New Castle, Del.

1894. Bliss, Arthur Ames, M.D., Laryngologist to the German Hospital; Laryngologist and Aurist to the Pennsylvania Institution for the Deaf and Dumb.

*1867. Boardman, Charles H., M.D., Boston, Mass.

1894. Bochroch, Max H., Instructor in Electro-Therapeutics and Chief Clinical Assistant in the Nervous Department, Jefferson Medical College Hospital.

1859. Boker, Charles S., M.D.

1891. Boyd, George M., M.D., Physician to the Philadelphia Lying-in Charity; Surgeon to the Out-door Department, Episcopal Hospital; Assistant Surgeon to the Kensington Hospital for Women.

†1884. Bradford, Thomas Hewson, M.D., Physician to the Dispensary of the Children's Hospital and to the Gynecological Department of the Pennsylvania Hospital.

1856. Brinton, John H., M.D., Professor of the Practice of Surgery and of Clinical Surgery in the Jefferson Medical College; Consulting Surgeon to the Southwestern Hospital of Philadelphia.

1891. Brinton, Lewis, M.D., Visiting Physician to the Nervous Department of Howard Hospital.

1887. Brubaker, Albert P., M.D., Professor of Physiology and General Pathology in the Pennsylvania College of Dental Surgery; Demonstrator of Physiology in Jefferson Medical College; Lecturer on Anatomy and Physiology at the Drexel Institute.

*1890. Brush, Edward N., M.D., Medical Superintendent of the Sheppard Asylum, Towson, Md.

*1851. Bullock, William R., M.D., Wilmington, Del.

ELECTED

1887. Bunting, Ross R., M.D.

1870. Burnett, Charles H., M.D., Professor (Emeritus) of Otology in the Philadelphia Polyclinic; Clinical Professor of Otology in the Woman's Medical College; Aural Surgeon, Presbyterian Hospital; and Consulting Aurist to Pennsylvania Institution for the Deaf and Dumb, West Philadelphia Hospital for Women, and the Bryn Mawr Hospital.

1892. Burr, Charles W., M.D., Visiting Physician to St. Joseph's Hospital and to the Home for Incurables; Visiting Pathologist to the State Asylum for the Insane.

1886. Cadwalader, Charles E., M.D.

1892. Cattell, Henry W., M.D., Demonstrator of Morbid Anatomy in the University of Pennsylvania; Pathologist to the Presbyterian Hospital.

*1892. Cerna, David, M.D., Ph.D., Demonstrator of Physiology in the Department of Medicine of the University of Texas; Corresponding Fellow of the Sociedad Española de Higiene of Madrid; Galveston, Texas.

1885. Chapin, John B., M.D., Physician to the Pennsylvania Hospital for the Insane.

1880. Chapman, Henry C., M.D., Professor of the Institutes of Medicine and of Medical Jurisprudence in Jefferson Medical College.

1868. Cheston, D. Murray, M.D.

1873. Clark, Leonardo S., M.D.

1872. Cleeman, Richard A., M.D.

*1842. Clymer, Meredith, M.D., New York.

1871. Cohen, J. Solis, M.D., Professor (Emeritus) of Diseases of the Throat and Chest in the Philadelphia Polyclinic; Professor (Honorary) of Laryngology in Jefferson Medical College; Consulting Physician to the Home for Consumptives, Philadelphia.

1888. Cohen, Solomon Solis, M.D., Professor of Clinical Medicine and Applied Therapeutics in the Philadelphia

ELECTED

Polyclinic, and Physician to the Polyclinic Hospital; Clinical Lecturer on Medicine in Jefferson Medical College; Visiting Physician to the Philadelphia Hospital; Consulting Physician to the Jewish Hospital.

1866. Cruice, R. B., M.D., Surgeon to St. Joseph's Hospital.

1884. Curtin, Roland G., M.D., Lecturer on Physical Diagnosis in the University of Pennsylvania; Assistant Physician to the University Hospital; Physician to the Philadelphia and Presbyterian Hospitals.

1884. Da Costa, John C., M.D., Gynecologist to Jefferson Medical College Hospital and to St. Agnes's Hospital.

†1858. Da Costa, J. M., M.D., LL.D., Professor (Emeritus) of the Principles and Practice of Medicine in Jefferson Medical College; Physician to the Pennsylvania Hospital; Consulting Physician to the Children's Hospital and to the Northern Dispensary.

1887. Daland, Judson, M.D., Instructor in Clinical Medicine and Lecturer on Physical Diagnosis in the University of Pennsylvania; Assistant Physician to the Hospital of the University of Pennsylvania; Physician to the Philadelphia Hospital; Editor-in-chief of the *International Clinics*.

1859. Darrach, James, M.D., Consulting Surgeon to the Germantown Hospital.

1888. Davis, Edward P., M.D., Professor of Obstetrics and Diseases of Infancy in the Philadelphia Polyclinic; Clinical Professor of Obstetrics in the Jefferson Medical College; Clinical Professor of Pædiatrics in the Woman's Medical College; Visiting Obstetrician to the Philadelphia Hospital; Physician to the Children's Department of the Howard Hospital; Member of the American Gynecological Society and of the American Pædiatric Society.

1889. Davis, G. G., M.D., M.R.C.S. Eng., Surgeon to the German and St. Joseph's Hospitals; Assistant Surgeon to the Orthopædic Hospital; Assistant Demonstrator of Surgery in the University of Pennsylvania.

ELECTED

1874. Deakyne, A. C., M.D.

1894. Deaver, Henry C., M.D., Surgeon to the Episcopal and St. Mary's Hospitals and to St. Christopher's Hospital for Children; Assistant Demonstrator of Anatomy, University of Pennsylvania.

1887. Deaver, John B., M.D., Professor of Surgery in the Philadelphia Polyclinic: Assistant Professor of Surgical Anatomy in the University of Pennsylvania; Attending Surgeon to the German and Philadelphia Hospitals; Consulting Surgeon to St. Agnes's, Germantown, and St. Timothy's Hospitals.

1892. Deaver, Richard Wilmot, M.D.

1885. Dercum, Francis X., M.D., Clinical Professor of Neurology in Jefferson Medical College; Neurologist to the Philadelphia Hospital.

1891. Dixon, Samuel G., M.D., Professor of Microscopic Technology and Histology, and Curator of the Academy of Natural Sciences of Philadelphia.

1891. Dixon, William C., M.D., Physician to Industrial Home for Blind Women, Philadelphia; Physician to the Shelter for Colored Orphans, Philadelphia; Member of Consulting Staff, Philadelphia Home for Incurables; Examiner of Insane Patients, Philadelphia Hospital.

1893. Downs, Norton, M.D.

1884. Downs, R. N., M.D.

1884. Drysdale, T. M., M.D.

1864. Duer, Edward L., M.D., Accoucheur to the Philadelphia Hospital; Surgeon to the Maternity Hospital; Visiting Physician to the Preston Retreat.

1871. Duhring, L. A., M.D., Professor of Skin Diseases in the University of Pennsylvania.

1881. Dulles, Charles Winslow, M.D., Surgeon to Rush Hospital; Surgeon to Out-patients, Presbyterian Hospital; Lecturer on History of Medicine, University of Penna.

1863. Dunglison, Richard J., M.D.

*1871. Dunglison, Thomas R., M.D., Paris, France.

ELECTED

1888. Dunn, Thomas D., M.D.

*1849. Dunnott, Justus, M.D., Harrisburg, Pa.

1860. Dunton, William R., M.D., Consulting Physician to the Germantown Hospital.

*1882. Edwards, Joseph F., M.D., Atlantic City, N. J.

*1887. Edwards, William A., M.D., San Diego, California.

1893. Eshner, Augustus A., M.D., Adjunct Professor of Clinical Medicine in the Philadelphia Polyclinic; Registrar in the Neurological Department of the Philadelphia Hospital.

*1880. Eskridge, J. T., M.D., Denver, Colorado.

1868. Evans, Horace Y., M.D., Physician to the Charity Hospital.

1894. Faries, Randolph, M.D., Surgeon to Orthopædic Dispensary in the University of Pennsylvania; Director of Physical Education, University of Pennsylvania and Protestant Episcopal Academy, Philadelphia.

1893. Farr, William W., M.D., Surgeon to Out-patient Department of the Episcopal Hospital; Visiting Physician to the Sheltering Arms and to the Church Home for Children.

1884. Fenton, Thomas H., M.D.

1866. Fischer, Emil, M.D.

1884. Fisher, Henry M., M.D., Physician to the Episcopal Hospital; Microscopist to the Pennsylvania Hospital, and Physician to the Out-patient Department of the same.

1888. Flick, Lawrence F., M.D.

1862. Forbes, William S., M.D., Professor of Anatomy in Jefferson Medical College.

1870. Ford, William H., M.D., President of the Board of Health of Philadelphia; Physician to the Foster Home.

†1885. Fox, Joseph M., M.D., Leesburg, Va.

1890. Freeman, Walter J., M.D., Adjunct Professor of Laryngology in the Philadelphia Polyclinic; Laryn-

ELECTED

gologist to the Out-patient Department of the Children's Hospital; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb.

1885. Fricke, Albert, M.D.

1893. Friebis, George, M.D., Ophthalmic Surgeon to the Mary Drexel Home and to the German Hospital.

1889. Fussell, M. Howard, M.D., Chief Physician to the Medical Dispensary of the University of Pennsylvania; Instructor of Clinical Medicine in the University of Pennsylvania.

1873. Gerhard, George S., M.D.

1884. Getchell, F. H., M.D.

1892. Gibb, Joseph S., M.D, Instructor in Diseases of Throat and Nose in the Philadelphia Polyclinic; Surgeon to the Ear, Nose, and Throat Department of the Episcopal Hospital.

1894. Gillespie, John, M.D., one of the Physicians to the Children's Hospital.

1885. Girvin, Robert M., M.D., Gynecologist to the Presbyterian Hospital.

1889. Githens, William H. H., M.D., Visiting Physician to the Sheltering Arms.

1894. Gleason, E. Baldwin, M.D., Clinical Professor of Otology in the Medico-Chirurgical College; Surgeon-in-charge of the Nose, Throat, and Ear Department, Northern Dispensary.

*1893. Gobrecht, William, M.D., Washington, D. C.

1884. Godey, Harry, M.D.

1893. Goodell, W. Constantine, M.D., Clinical Instructor of Gynecology in the University of Pennsylvania; Assistant Gynecologist, University Hospital.

1867. Goodman, H. Ernest, M.D., Professor of Surgery in the Medico-Chirurgical College; Surgeon to Wills Eye Hospital and to the Orthopædic Hospital; Consulting Surgeon to the Maternity Hospital.

ELECTED

1894. Graham, Edwin E., M.D., Clinical Professor of Diseases of Children in the Jefferson Medical College; Physician to the Franklin Reformatory Home.

1885. Graham, John, M.D.

1891. Green, Walter D., A.M., M.D., Out-patient Surgeon to the Pennsylvania, Children's, and Methodist Hospitals; Assistant Surgeon to the Gynecean Hospital.

1870. Grier, M. J., M.D.

1883. Griffith, J. P. Crozer, M.D., Clinical Professor of the Diseases of Children in the Hospital of the University of Pennsylvania; Professor of Clinical Medicine in the Philadelphia Polyclinic; Physician to St. Agnes's, the Children's, the Howard, and the Rush Hospitals.

1871. Grove, John H., M.D., Surgeon to St. Mary's and to St. Agnes's Hospital.

1889. Guitéras, John, M.D., Professor of General Pathology and Morbid Anatomy in the University of Pennsylvania.

1893. Hamill, Robert H., M.D.

1894. Hamill, Samuel McC., Demonstrator of Physical Diagnosis, University of Pennsylvania; Assistant Physician to the Medical Dispensary of the Hospital of the University of Pennsylvania; Visiting Physician to the Baptist Orphanage.

*1859. Hammond, William A., M.D., Surgeon-General U. S. A. (Retired), Washington, D. C.

1886. Hansell, Howard F., M.D., Chief Clinical Assistant to the Ophthalmological Department of Jefferson Medical College Hospital; Ophthalmic and Aural Surgeon to the Southwestern Hospital.

1889. Hare, Hobart A., M.D., Professor of Therapeutics in Jefferson Medical College; Physician to St. Agnes's Hospital and to Jefferson Medical College Hospital.

1865. Harlan, George C., M.D., Surgeon to Wills Eye Hospital and to the Eye and Ear Department of the Pennsylvania Hospital; Professor (Emeritus) of Diseases of the Eye in the Philadelphia Polyclinic.

ELECTED

1863. Harlow, Lewis D., M.D.

1862. Harris, Robert P., M.D.

1885. Harte, Richard H., M.D., Surgeon to Pennsylvania Hospital; Surgeon to Episcopal Hospital; Surgeon to St. Mary's Hospital; Consulting Surgeon to St. Timothy's Hospital; Demonstrator of Osteology, University of Pennsylvania.

1851. Hartshorne, Henry, M.D., LL.D.

1888. Hartzell, Milton B., M.D., Instructor in Dermatology, University of Pennsylvania.

1872. Hays, I. Minis, M.D.

1882. Hearn, W. Joseph, M.D., Surgeon to the Hospital of Jefferson Medical College and to the Philadelphia Hospital.

1884. Henry, Frederick P., M.D., Physician to Jefferson Medical College Hospital and to the Philadelphia Hospital; Professor of the Principles and Practice of Medicine in the Woman's Medical College of Pennsylvania.

1891. Hewson, Addinell, A.M., M.D., Demonstrator of Anatomy in Jefferson Medical College; Dispensary Surgeon to the Hospital of the Protestant Episcopal Church.

1872. Hinkle, A. G. B., M.D.

1892. Hinsdale, Guy, M.D, Lecturer on Climatology in the University of Pennsylvania; Physician to the Presbyterian Orphanage and to the Out-patient Department of the Presbyterian Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases.

1888. Hirsh, Abram B., M.D.

1888. Hirst, Barton Cooke, M.D., Professor of Obstetrics in University of Pennsylvania; Obstetrician to the Philadelphia Hospital and to the Maternity Hospital.

1894. Hoch, William R., M.D., Laryngologist to Rush Hospital; Assistant Physician in the Dispensary for Diseases of the Throat and Nose, Hospital of the University of Pennsylvania.

ELECTED

1885. Holland, James W., M.D., Professor of Medical Chemistry and Toxicology in Jefferson Medical College.

†1879. Hopkins, William Barton, M.D., Senior Surgeon to the Episcopal Hospital and to the Orthopædic Department of the Pennsylvania Hospital.

1867. Horn, George H., M.D., Professor of Entomology in the Biological Department of the University of Pennsylvania.

1888. Horwitz, Orville, M.D., Clinical Professor of Genito-urinary Diseases in Jefferson Medical College; Professor of Genito-urinary Surgery in the Philadelphia Polyclinic, and Surgeon to the Philadelphia Hospital.

1868. Howell, Samuel B., M.D., Professor of Chemistry in the Medico-Chirurgical College.

1892. Hughes, William E., M.D.

°1854. Hunt, William, M.D., Surgeon to the Pennsylvania Hospital.

1871. Ingham, James V., M.D.

1885. Jackson, Edward, M.D., Professor of Diseases of the Eye in the Philadelphia Polyclinic; Surgeon to Wills Eye Hospital; Ophthalmologist to Rush Hospital.

1887. Jayne, Horace, M.D., Professor of Vertebrate Morphology in the Biological Department of the University of Pennsylvania.

1885. Judd, Leonardo Da Vinci, M.D.

1867. Judson, Oliver A., M.D.

1886. Jurist, Louis, M.D., Chief Clinical Assistant in the Laryngological Department of Jefferson Medical College Hospital.

†1867. Keen, William W., M.D., LL.D, Professor of the Principles of Surgery and of Clinical Surgery in Jefferson Medical College; Surgeon to Jefferson Medical College Hospital and to the Orthopædic Hospital and Infirmary for Nervous Diseases, and Consulting Surgeon to St.

ELECTED

Agnes's Hospital and to the Woman's Hospital; Membre Correspondant Étranger de la Société de Chirurgie de Paris.

*1887. Kelly, Howard A., M.D., Professor of Gynecology in the Johns Hopkins University, and Gynecologist and Obstetrician to the Hospital, Baltimore, Md.

*1844. King, Charles R., M.D., Andalusia, Pa.

†1875. Kirkbride, Joseph J., M.D.

1892. Lainé, Damaso T., M.D.

*1865. La Roche, C. Percy, M.D., Rome, Italy.

1887. Leaman, Henry, M.D.

1893. LeConte, Robert G., M.D., Surgeon to Out-patient Departments of the Pennsylvania Hospital, Children's Hospital, and Methodist Episcopal Hospital; Assistant Surgeon to the Gynecean Hospital; Assistant Surgeon to the Genito-urinary Department of the University of Pennsylvania; Assistant Surgeon, 3d Regiment, N. G. of Pa.

1883. Leffmann, Henry, M.D., Professor of Chemistry in the Philadelphia Polyclinic and in the Woman's Medical College; Pathological Chemist to Jefferson Medical College Hospital.

1892. Leidy, Joseph, M.D., Physician to Out-patient Department of the Pennsylvania Hospital; Assistant Surgeon to the Genito-urinary Department of the University of Pennsylvania.

1855. Lewis, Francis W., M.D.

1877. Lewis, Morris J., M.D., Physician to the Children's Hospital, to the Orthopædic Hospital and Infirmary for Nervous Diseases, and to the Pennsylvania Hospital.

1886. Lloyd, J. Hendrie, M.D., Physician to the Nervous and Insane Department of the Philadelphia Hospital, to the Methodist Episcopal Hospital, and to the Home for Crippled Children.

ELECTED

1893. Longaker, Daniel, M.D.

1877. Longstreth, Morris, M.D., Professor of Pathological Anatomy in Jefferson Medical College; Physician to the Pennsylvania Hospital.

1886. MacCoy, Alexander W., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic; Lecturer on Diseases of the Throat and Nose in the Woman's Medical College of Pennsylvania.

1875. McClellan, George, M.D., Surgeon to the Howard Hospital.

1871. McFerran, J. A., M.D.

*1885. Mallet, John W., M.D., Charlottesville, Va.

1893. Marshall, John, M.D., Assistant Professor of Chemistry in the University of Pennsylvania.

1889. Martin, Edward, M.D., Surgeon to the Howard Hospital; Clinical Professor of Genito-urinary Diseases in the University of Pennsylvania.

1887. Massey, Isaac, M.D., Surgeon to the Pennsylvania Railroad; Physician to the Chester County Hospital.

*1850. Mayer, Edward R., M.D., Wilkesbarre, Pa.

1885. Mays, Thomas J., M.D., Professor of Diseases of the Chest and of Experimental Therapeutics in the Philadelphia Polyclinic; Visiting Physician to Rush Hospital.

1868. Mears, J. Ewing, M D., Professor of Anatomy and Clinical Surgery in the Pennsylvania College of Dental Surgery; Gynecologist to Jefferson Medical College Hospital; Surgeon to St. Agnes's Hospital.

1875. Meigs, Arthur V., M.D., Physician to the Pennsylvania Hospital; Consulting Physician to the Pennsylvania Institution for the Instruction of the Blind.

*1884. Mifflin, Houston, M.D., Columbia, Pa.

1894. Miller, D. J. Milton, M.D., Physician to the Episcopal Hospital; Assistant Physician to the Children's Hospital.

1881. Mills, Charles K., M.D., Professor of Mental Diseases and of Medical Jurisprudence in the University of Penn-

ELECTED

sylvania; Professor of Diseases of the Mind and Nervous System in the Philadelphia Polyclinic; Clinical Professor of Nervous Diseases in the Woman's Medical College; Neurologist to the Philadelphia Hospital.

†1888. Mitchell, John K., M.D., Instructor in Clinical Medicine in the University of Pennsylvania; Physician to St. Agnes's Hospital; Assistant Physician to the University Hospital and to the Infirmary for Nervous Diseases.

1856. Mitchell, S. Weir, M.D., Professor of Diseases of the Mind and Nervous System in the Philadelphia Polyclinic; Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Consulting Physician to the Maternity Hospital.

1882. Montgomery, Edward E., M.D., Clinical Professor of Gynecology in the Jefferson Medical College; Obstetrician to the Philadelphia Hospital.

1863. Morehouse, George R., M.D., Ph.D.

1886. Morris, Caspar, M.D., Physician to the Episcopal Hospital and to the Out-patient Department of the Pennsylvania Hospital.

1893. Morris, Elliston J., M.D.

1883. Morris, Henry, M.D., Gynecologist to the Howard Hospital.

1856. Morris, J. Cheston, M.D.

1861. Morton, Thomas G., M.D., Surgeon to the Pennsylvania and the Orthopædic Hospitals; Consulting Surgeon to the Jewish Hospital; Emeritus Surgeon to Wills Eye Hospital.

1891. Morton, T. S. K., M.D., Professor of Surgery in the Philadelphia Polyclinic; Surgeon to the Polyclinic Hospital; Assistant Surgeon to the Orthopædic Hospital; Surgeon to Out-patient Department of the Pennsylvania Hospital. Consulting Surgeon to the Philadelphia Dispensary.

1864. Moss, William, M.D.

1890. Müller, Auguste F., M.D., Attending Physician to the Germantown Hospital.

ELECTED

1882. Musser, John H., M.D., Assistant Professor of Clinical Medicine in the University of Pennsylvania; Physician to the Philadelphia Hospital and to the Presbyterian Hospital; Consulting Physician to the Woman's Hospital of Philadelphia and to the West Philadelphia Hospital for Women.

1886. Neff, Joseph F., M.D.

1887. Neilson, Thomas Rundle, M.D., Surgeon to the Episcopal Hospital and to St. Christopher's Hospital for Children; Professor of Genito-urinary Diseases in the Philadelphia Polyclinic; Lecturer on Diseases of the Rectum and Assistant Demonstrator of Anatomy in the University of Pennsylvania.

1889. Noble, Charles P., M.D., Surgeon-in-Chief to the Kensington Hospital for Women; Surgeon-in-Charge of the Department for Women of the Northern Dispensary; Surgeon-in-Charge of the Department for Women of the Union Dispensary; Lecturer on Gynecology in the Philadelphia Polyclinic.

1893. Noble, William H., M.D.

1869. Norris, Herbert, M.D., Supervising Physician to St. Clement's Hospital.

1865. Norris, Isaac, Jr., M.D.

1892. Norris, Richard C., M.D., Demonstrator of Obstetrics, University of Pennsylvania; Assistant Obstetrician, University Maternity; Obstetric Registrar, Philadelphia Hospital; Visiting Physician to the Methodist Hospital; Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary.

1866. Norris, William F., M.D., Honorary Professor of Ophthalmology and Clinical Professor of Diseases of the Eye in the University of Pennsylvania; Surgeon to Wills Eye Hospital.

1884. Oliver, Charles A., M.D., Attending Surgeon to Wills Eye Hospital; Ophthalmic Surgeon to the Philadelphia

ELECTED

and the Presbyterian Hospitals; Consulting Ophthalmic Surgeon to St. Mary's, St. Agnes's, St. Timothy's, and Maternity Hospitals.

1884. O'Neill, J. W., M.D.

*1885. Osler, William, M.D., Professor of Medicine in Johns Hopkins University, and Physician to the Hospital, Baltimore, Md.

1890. Packard, Frederick A., M.D., Visiting Physician to the Episcopal and Methodist Hospitals; Physician to the Out-patient Department of the Pennsylvania and Children's Hospitals; Instructor in Physical Diagnosis in the University of Pennsylvania.

1858. Packard, John H., M.D., Surgeon to the Pennsylvania Hospital and to St. Joseph's Hospital.

1864. Pancoast, William H., M.D., Professor of Anatomy and of Clinical Surgery in the Medico-Chirurgical College; Consulting Surgeon to the Philadelphia Hospital for Skin Disease.

1882. Parish, William H., M.D., Professor of Obstetrics in the Dartmouth Medical College; Professor of Anatomy in the Woman's Medical College of Pennsylvania; Consulting Obstetrician to the Lying-in Charity; Consulting Surgeon to the Kensington Hospital; Consulting Gynecologist to St. Agnes's Hospital.

1883. Parvin, Theophilus, M.D., Professor of Obstetrics and Diseases of Women and Children in the Jefferson Medical College.

†1889. Penrose, Charles Bingham, M.D., Professor of Gynecology in the University of Pennsylvania; Surgeon to the Gynecean Hospital.

1854. Penrose, R. A. F., M.D., LL.D., Professor (Emeritus) of Obstetrics and Diseases of Women and Children in the University of Pennsylvania; Consulting Obstetrician to the Maternity Hospital; Visiting Physician to the Preston Retreat.

ELECTED

1868. Pepper, William, M.D., LL.D., Provost of the University of Pennsylvania, and Professor of the Theory and Practice of Medicine in the same.

1884. Perkins, Francis M., M.D., Ophthalmic Surgeon to St. Agnes's Hospital; Ophthalmic Surgeon to the Presbyterian Hospital.

1890. Phillips, J. Willoughby, M.D.

1883. Piersol, George A., M.D., Professor of Anatomy in the University of Pennsylvania.

1872. Porter, William G., M.D., Surgeon to the Presbyterian Hospital and to the Philadelphia Hospital.

1885. Potter, Thomas C., M.D.

1887. Price, Jacob, M.D.

†1889. Price, Joseph, M.D., Obstetric Physician to the Philadelphia Dispensary.

1889. Randall, B. Alexander, M.D., Professor of Otology in the University of Pennsylvania and in the Philadelphia Polyclinic; Ophthalmic and Aural Surgeon to the Children's Hospital; Otologist to Rush Hospital.

1887. Reed, Charles H., M.D.

1885. Reichert, Edward T., M.D., Professor of Physiology in the University of Pennsylvania.

1888. Rex, George A., M.D.

1891. Rhoads, Edward G., M.D.

1891. Risley, S. D., M.D., Lecturer on Ophthalmology in the University of Pennsylvania; Attending Surgeon at the Wills Eye Hospital; Professor of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine.

1882. Roberts, A. Sydney, M.D.

†1878. Roberts, John B., M.D., Professor of Anatomy and Surgery in the Philadelphia Polyclinic; Professor of Surgery in the Woman's Medical College of Pennsylvania; Surgeon to the Methodist Hospital.

ELECTED

1888. Robins, Robert P., M.D., Visiting Physician to the Dispensary of the House of Industry, to the Church Home for Children, and to the Board of Guardians of the Poor; Lecturer on Chemistry in the Episcopal Academy.

†1838. Ruschenberger, W. S. W., M.D., Medical Director, U. S. Navy.

*1864. Sargent, Winthrop, M D., Roxbury, Mass.

†1866. Schaffer, Charles, M.D., Professor of Botany in the Pennsylvania Horticultural Society.

1887. De Schweinitz, George E., M.D., Clinical Professor of Ophthalmology in Jefferson Medical College; Professor of Ophthalmology in the Philadelphia Polyclinic; Ophthalmic and Aural Surgeon to the Children's Hospital; Ophthalmologist to the Orthopædic Hospital and to the Philadelphia Hospital; Consulting Ophthalmic Surgeon to the Methodist Episcopal Hospital.

1892. Seiss, Ralph W., M.D., Professor of Otology in the Philadelphia Polyclinic.

1888. Seltzer, Charles M., M.D.

1875. Seyfert, Theodore H., M.D.

1884. Shaffner, Charles, M.D., Ophthalmic Surgeon to the Presbyterian Hospital.

1887. Shakespeare, Edward O., M.D., Pathologist to the Philadelphia Hospital.

1876. Shippen, Edward, A.M., M.D., U. S. Navy (retired).

1891. Shober, John B., M.D., Surgeon to the University Hospital Dispensary and to the Gynecean Hospital Dispensary.

1890. Shoemaker, George Erety, A.M., M.D., Gynecologist to the Methodist Hospital and to the St. Clement's Hospital for Epilepties.

†1893. Shoemaker, Harvey, M.D., Visiting Physician to the Sheltering Arms; Physician to Out-patient Department of the German, Episcopal, and Children's Hospitals.

ELECTED

1880. Simes, J. H. C., M.D., Professor of Genito-urinary and Venereal Diseases in the Philadelphia Polyclinic; Surgeon to St. Christopher's Hospital.

1873. Simpson, James, M.D.

1872. Sinkler, Wharton, M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Neurologist to the Philadelphia Hospital; Physician to the Epileptic Hospital of Philadelphia.

*1863. Smith, A. K., M.D., U. S. A. (retired), New Hartford, Conn.

*1864. Smith, Edward A., M.D., New York.

1894. Stahl, B. Franklin, Ph.G., B.S., M.D., Instructor in Physical Diagnosis in the University of Pennsylvania; Visiting Physician to St. Agnes's Hospital; Ophthalmic Surgeon to Charity Hospital.

1875. Starr, Louis, M.D., Physician to the Children's Hospital; Consulting Physician to the Maternity Hospital.

1892. Steinbach, Lewis W., M.D., Professor of Clinical and Operative Surgery in the Philadelphia Polyclinic; Visiting Surgeon to the Philadelphia Hospital and to the Jewish Hospital of Philadelphia.

1884. Stelwagon, Henry W., M.D, Ph.D., Clinical Professor of Dermatology in Jefferson Medical College; Dermatologist to the Philadelphia Hospital; Physician to the Department for Skin Diseases of the Howard Hospital and the Northern Dispensary; Clinical Professor of Dermatology in the Woman's Medical College.

1888. Stewart, David D., M.D., Clinical Lecturer on Medicine in Jefferson Medical College; Physician to St. Mary's Hospital and to St. Christopher's Hospital for Children.

†1842. Stillé, Alfred, M.D., LL.D., Professor (Emeritus) of the Theory and Practice of Medicine in the University of Pennsylvania; Consulting Physician to the Maternity Hospital and to the Woman's Hospital.

°1846. Stocker, Anthony E., M.D.

ELECTED

1884. Stryker, Samuel S., M.D., Physician to the Presbyterian Hospital.

1866. Taylor, John Madison, M.D., Neurologist to Howard Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Assistant Physician to the Children's Hospital; Professor of Children's Diseases in the Philadelphia Polyclinic; Fellow of the American Academy of Medicine.

1867. Taylor, R. R., M.D.

1887. Taylor, William J., M.D., Surgeon to St. Agnes's Hospital, and Assistant Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases; Professor of Orthopædic Surgery in the Philadelphia Polyclinic.

1886. Taylor, William L., M.D., Instructor in Clinical Gynecology in the University of Pennsylvania, and Chief of the Clinic and Assistant Gynecologist to the Hospital of the same; Surgeon-in-Chief to the Beacon Service for Women.

1867. Thomas, Charles H., M.D.

†1869. Thomson, William, M.D., Professor (Honorary) of Ophthalmology in the Jefferson Medical College, and Ophthalmic Surgeon to the Hospital of the same; Emeritus Surgeon to Wills Eye Hospital.

1894. Tunis, Joseph Price, M.D., Assistant Demonstrator of Anatomy and Surgery in the University of Pennsylvania; Surgeon to the Dispensaries of the Presbyterian, Methodist, Episcopal, and Children's Hospitals.

1866. Tyson, James, M.D., Professor of Clinical Medicine in the University of Pennsylvania; Physician to the University Hospital; Visiting Physician to the Philadelphia Hospital.

1864. Vandyke, E. B., M.D.

1873. Van Harlingen, Arthur, M.D., Professor of Diseases of the Skin in the Philadelphia Polyclinic; Dermatologist to the Howard Hospital.

ELECTED

1893. Vansant, Eugene Larue, M.D., Lecturer on Clinical Medicine in Jefferson Medical College; Visiting Physician to the Philadelphia Hospital.

1883. Vinton, Charles Harrod, M.D.

1885. Walker, James B., M.D., Attending Physician to the Philadelphia Hospital; Lecturer on Clinical Medicine and Consulting Physician to the Woman's Hospital.

1893. Warren, Joseph W., M.D., Associate Professor of Physiology in Bryn Mawr College.

1886. Watson, E. W., M.D., Physician to the Pennsylvania Institution for the Blind; Visiting Physician to the Home for Consumptives.

1875. Webb, William H., M.D.

1883. Welch, William M., M.D., Physician in Charge of the Municipal Hospital for Contagious Diseases; Consulting Physician to the Northern Dispensary; Consulting Physician to the Northern Home for Friendless Children.

1893. Westcott, Thompson S., M.D., Visiting Physician to the Methodist Episcopal Hospital; Physician to Dispensary for Diseases of Children, University of Pennsylvania.

1884. Wharton, Henry R., M.D., Demonstrator of Surgery in the University of Pennsylvania, and Assistant Surgeon to the Hospital of the University of Pennsylvania; Surgeon to the Children's, Presbyterian, and Methodist Hospitals.

1883. Whelen, Alfred, M.D.

1878. White, J. William, M.D., Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the Maternity Hospital.

†1880. Willard, De Forest, M.D., Clinical Professor of Orthopædic Surgery in the University of Pennsylvania; Surgeon to the Presbyterian Hospital; Consulting Surgeon to the White and to the Colored Cripples' Homes and to the Home for Incurables.

*1878. Williamson, Jesse, M.D., Colorado Springs, Colorado.

ELECTED

1881. Wilson, H. Augustus, M.D., Professor of General and Orthopædic Surgery in the Philadelphia Polyclinic; Clinical Professor of Orthopædic Surgery in the Jefferson Medical College; Consulting Orthopædic Surgeon to the Philadelphia Lying-in Charity and to the Kensington Hospital for Women.

1874. Wilson, James C., M.D., Professor of the Practice of Medicine and of Clinical Medicine in Jefferson Medical College, and Physician to the Hospital of the same (Faculty Staff); Physician to the German Hospital.

†1884. Wirgman, Charles, M.D., Physician to the Hospital of Jefferson Medical College and to the Howard Hospital; Physician to Out-patient Department of Children's Hospital.

1852. Wister, Owen Jones, M.D., Consulting Physician to the Jewish Hospital.

1893. Wolff, Lawrence, M.D., Demonstrator of Chemistry in the Jefferson Medical College; Visiting Physician to the German Hospital; Clinical Professor of Medicine in the Woman's Medical College.

1893. Wood, Alfred C., M.D., Assistant Surgeon to Gynecean Hospital; Instructor in Clinical Surgery in the University of Pennsylvania; Assistant Surgeon to the University Hospital; Surgeon to Out-patient Department of the University Hospital.

1865. Wood, Horatio C., M.D., Professor of Materia Medica, Pharmacy, and General Therapeutics in the University of Pennsylvania, and Clinical Professor of Diseases of the Nervous System in the Hospital of the same.

1880. Woodbury, Frank, M.D., Honorary Professor of Clinical Medicine in the Medico-Chirurgical College of Philadelphia, and Physician to the Hospital of the same.

1866. Woods, D. F., M.D., Physician to the Presbyterian Hospital.

1888. Woodward, Charles E., M.D., Physician to the Chester County Prison and West Chester Board of Health; U. S. Examining Surgeon.

ELECTED

1878. Wormley, Theodore G., M.D., LL.D., Professor of Chemistry in the University of Pennsylvania.

1860. Wurts, Charles Stewart, M.D., Physician to Foster Home.

1861. Yarrow, Thomas J., M.D.

1889. Young, James K., M.D., Instructor in Orthopædic Surgery and Assistant Demonstrator of Surgery in the University of Pennsylvania; Orthopædic Surgeon in the Out-patient Department of the Hospital.

1894. Zentmayer, William, M.D., Assistant Surgeon to Wills Eye Hospital; Ophthalmologist to St. Mary's Hospital, Philadelphia, and to the Stratford Castle Hospital, Phœnixville, Pa.

1887. Ziegler, Walter M. L., M.D., Assistant Aural Surgeon and Chief of the Dispensary for Diseases of the Ear in the Hospital of the University of Pennsylvania.

ASSOCIATE FELLOWS.

[Limited to Fifty, of whom Twenty may be Foreigners.]

ELECTED

1873. Acland, Henry W., M.D., F.R.S., Oxford, England.
1890. Baccelli, Guido. Rome, Italy.
1877. Barnes, Robert, M.D., London, England.
1876. Billings, John S., M.D., U. S. A., Washington, D. C.
1886. Bowditch, Henry P., M.D., Boston, Massachusetts.
1894. Brunton, T. Lauder, M.D., London, England.
1877. Chaillé, Stanford E., M.D., New Orleans, Louisiana.
1886. Cheever, David W., M.D., Boston, Massachusetts.
1876. Comegys, C. G., M.D., Cincinnati, Ohio.
1876. Corson, Hiram, M.D., Norristown, Pennsylvania.
1893. Councilman, William T., M.D., Boston, Mass.
1876. Davis, N. S., M.D., Chicago, Illinois.
1886. Draper, William H., M.D., New York.
1892. Emmet, Thomas Addis, M.D., New York.
1883. Fayrer, Sir Joseph, M.D., LL.D., F.R.S., London, England.
1892. Fitz, Reginald H., M.D., Boston, Mass.
1876. Green, Traill, M.D., Easton, Pennsylvania.
1883. Heath, Christopher, F.R.C.S., London, England.
1874. Jackson, J. Hughlings, M.D., London, England.
1891. Jacobi, A., M.D., New York.
1893. v. Jaksch, Rudolf, M.D., Prague, Bohemia.
1876. Johnson, Sir George, M.D., F.R.S., London, England.
1876. Jones, Joseph, M.D., New Orleans, Louisiana.
1893. Kerr, John G., M.D., Canton, China.
1877. Lister, Sir Joseph, Bart., M.D., LL.D., F.R.S., London, England.

ELECTED

1886. McGuire, Hunter, M.D., Richmond, Virginia.
1876. Moore, E. M., M.D., Rochester, New York.
1876. Mowry, R. B., M.D., Allegheny City, Pennsylvania.
1873. Ogle, John W., M.D., London, England.
1874. Paget, Sir James, Bart., M.D., LL.D., F.R.S., D.C.L., London, England.
1876. Pollock, A. M., M.D., Pittsburg, Pennsylvania.
1876. Porcher, F. Peyre, M.D., Charleston, South Carolina.
1886. Reeve, John C., M.D., Dayton, Ohio.
1886. Senn, Nicholas, M.D., Milwaukee, Wisconsin.
1886. Thomas, T. Gaillard, M.D., New York.
1869. Valcourt, Th. de, M.D., Cannes, France.
1892. Virchow, Rudolf, M.D., Berlin, Germany.
1894. Warren, J. Collins, M.D., Boston, Mass.
1894. Weir, Robert F., M.D., New York.
1892. Welch, William H., M.D., Baltimore, Maryland.
1886. Whittaker, James T., M.D., Cincinnati, Ohio.
1886. Yandell, David W., M.D., Louisville, Kentucky.

CORRESPONDING MEMBERS.

ELECTED

1880. Carrow, Flemming, M.D., United States.
1880. Chiara, Domenico, M.D., Florence, Italy.
1886. Dey, Kanny Lall, M.D., Calcutta, India.
1885. Rendu, Jean, M.D., Lyons, France.
1886. Richards, Vincent, Golunda, India.
1889. Strahan, John, M.D., Belfast, Ireland.

NECROLOGICAL LIST FOR 1894.

FELLOWS

WILLIAM V. KEATING	April 18, 1894.
WILLIAM GOODELL	October 27, 1894.

ASSOCIATE FELLOW

OLIVER WENDELL HOLMES	October 7, 1894.

RESIGNED FELLOWSHIP

A. DOUGLASS HALL	January 3, 1894.

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MEMOIR OF JOHN M. KEATING, M.D.

By JOHN K. MITCHELL, M.D.

[Read December 5, 1894.]

Dr. John M. Keating descended upon both sides from people who have helped to add distinction to our profession in Philadelphia. His great-grandfather emigrated from France, whither the Catholic troubles of the Seventeenth Century had driven the family from Ireland after the defeat of James II. by King William; Baron John Keating was a trustee of the University of Pennsylvania for many years; one of his sons was a professor in that institution; another, Dr. William V. Keating, graduated from the University, as did also his son, the subject of this memoir.

Dr. William V. Keating was Professor of Obstetrics in the Jefferson Medical College, succeeding Dr. C. D. Meigs in that chair. He married in 1851 the daughter of Dr. Renè La Roche, a graduate of the University and a Fellow of this College, whose observations of yellow fever in the first half of the century make a work on that disease which retains and will retain a place of distinguished importance in medical literature.

John M. Keating was born in Philadelphia in 1852, and went from the Polytechnic College to the University of Pennsylvania, where he graduated in medicine in 1873. A service as Resident Physician at the Philadelphia Hospital followed and began a career of unceasing medical industry which was ended only by his premature death last year. He held many medical appointments and wrote untiringly. He was Physician to

Blockley Hospital and Lecturer on the Diseases of Children in the University of Pennsylvania, thus taking up a subject in which previously his father had done good work; he was gynecologist to St. Joseph's Hospital, and fulfilled various duties at the St. Agnes', Howard, and Children's Hospitals, and was besides Medical Director of the Penn Mutual Life Insurance Company for ten years.

His activity in various medical societies, both local and national, was equally great. He was a Fellow of this College and of the American and British Gynecological Societies, and was at different times President of the Pediatric Society, of the Association of Life Insurance Medical Directors, and of the Section on Diseases of Children of the Pan-American Medical Congress. He edited the *Archives of Pediatrics*, the *International Clinics*, and, during its brief existence, *The Climatologist*, and was never without a busy interest in some work on hand, having written many medical and popular books, the latter mostly for the aid of mothers in the care of children.

His most important labor, and the one in which his own best showed, was the well-known *Cyclopædia of Diseases of Children*, in which he succeeded in associating with him many of the best known men of America and England, and produced a most valuable and representative book. Much of the success of such work necessarily depends upon the editor, not only in his first selection of his co-laborers, but in his management of them and of their contributions. Dr. Keating was well fitted in both of these requirements. He had a vast acquaintance with the medical men of both Europe and America. His constant and genial good humor was but one of the qualities which made him greatly liked by every one with whom he came in contact; he had excellent taste in the literary part of the matter and an altogether admirable tact in the ticklish business of suggesting such changes as he thought desirable in the articles of his contributors. The result, as has been said, was very good, and he received much well-deserved praise for it.

Some of the other books in which he had a hand were: *The*

Mother's Guide to the Management and Feeding of Infants; Mother and Child; Maternity, Infancy and Childhood; A Manual for Life Insurance Examiners; A New Dictionary of Medicine; Diseases of the Heart and Circulation in Children; several works on the climate of Florida and Colorado, and innumerable contributions to various medical journals, besides articles for Sajous's *Annual*, Buck's *Handbook*, and Pepper's *System*.

My own first acquaintance with Dr. Keating was when, soon after his service as Resident Physician at Blockley, I as a lad went in company with him and two older members of our profession on a fishing trip up the Nipigon River, in Canada, where his constant amiability, the facile pencil with which he sketched the scenery or the humors of our little expedition, the ready wit with which he amused us, the good nature with which he took a jest upon himself, in a word, his companionability, made him a great part of the life of the camp.

From that time until he broke down with the pulmonary trouble which finally caused his death I saw him constantly. It was soon after the publication of the *Cyclopædia* that he developed the alarming symptoms which, viewed in connection with an hereditary predisposition and a health always feeble, made his advisers insist upon his removal to the high altitudes of Colorado, where, although he improved in health, and resumed to some extent the practice of the profession in which he was so wholly wrapped up, he never recovered from a sense of exile and banishment. When the brief annual visits that he was able to make to his home came round, I remember he said more than once to me, if I asked him to come to a hospital or see some interesting case, that he could not go, for the very sight of a hospital or the too familiar odor of such an institution made him horribly homesick.

Work was in some sort his panacea for distress of mind or bodily pain; but still more he labored because he loved the work. He was, indeed, wholly absorbed by and devoted to his profession; no other intellectual questions ever attracted him as medical ones did; and perhaps this singleness of interest was both help and hurt—a help when health for the physical

effort of practice failed; a hurt because he still worked on when unable to do himself justice, and when, had he had a greater variety of other interests to turn to, he might have had solace and diversion at a less cost in expenditure of strength.

But no feebleness of body, no trouble of mind, was ever permitted to interfere with his constant industry or with his thoughtfulness for those he loved. His kindly, cordial manner, his humorous talk were always the same; and the courage with which he bore a progressive failure of health was unflinching; with the least hint of remission in oppressing or painful symptoms he would struggle back to his desk and his work again.

When we saw him last here he was better than for several years, and it seemed as if the enemy were finally defeated; but a cold, at first slight, developed into pneumonia, and he died on November 17, 1893, as bravely and calmly as he had lived and suffered.

Had his physical strength been as great as his activity of mind and his strenuous energy needed, he would have accomplished great things and left his mark upon the world. As it is, he has left us the happy memory of a faithful friend, a workman who did honestly whatever he set his hand to, and always the recollection of a brave, tender, and kindly gentleman.

ANNUAL ADDRESS OF THE PRESIDENT.

By S. WEIR MITCHELL, M.D.

[Read December 5, 1894.]

At this time, and near to the close of my second presidential term of office, it becomes my duty, in accordance with your by-law, to speak of matters connected with the College which seem to invite comment.

During my last presidential term the College work has so increased that the meetings have been overcrowded with papers. Interesting debates have been, of need, cut short to make time for private business—the consideration of new candidates, change in by-laws, and other matters of the most grave importance. Thus I have usually found that late in the night, with barely a quorum, business was too often hastily disposed of which, having been carefully matured in council and elsewhere, should have exacted more patient treatment.

To remedy this over-pressure of work, and for other reasons, I favored the creation of Sections. These, it was feared, would leave the College meetings bare of interest. It has not been so. We are still more than supplied with papers, and still find too little leisure for the general business of the College. We need further relief; and no matter how many Sections we may create, the more important papers and the great debates will surely seek audience at your general meetings. Yet, if at any time there should be lacking such material, it will be easy to supply it by asking a Section to fill for us here an evening, and to bring before us something in its specialty which has interesting relation to general medicine and to the work of the everyday practitioner.

You have now Sections of Ophthalmology, of Orthopedic Surgery, of General Surgery, and of Otology and Laryngology. I have lately authorized, under your ordinance, a Section of Gynecology, and I would add a Section of General Medicine, and thus further relieve the College meetings. My proposition has the approval of many of the Fellows.

The Library, the Museum, the Sections, and all other branches of our work will speak through the respective committees, all of which have to record satisfactory results.

The Fellowship has gained through the year 16 Fellows and 3 Associate Fellows. The College now numbers in all 289 Fellows and 42 Associate Fellows, of whom 15 are foreign and 27 American, also 6 corresponding members.

Despite our utmost efforts the Council has been often left without enough information concerning those whose names are presented for Fellowship. I have, therefore, proposed to the Council to ask of you permission to insist on the proposers of a candidate furnishing a statement of what places he has held and what papers he has published.

We have lost during the year one Fellow by resignation, and two, Dr. William Goodell and Dr. W. V. Keating, by death. Also, we have to mourn the loss of Oliver Wendell Holmes—an Associate Fellow. It so happens that with the two Fellows we have thus lost I was united by very close bonds of affectionate esteem, and I can speak of neither of them without such emotion as a man may feel who sees fall around him the friends of many years. Dr. Keating, at one time in large practice, was one of the many on our roll who took with him into all the varied relations of public and private life the charm of refinement and cultivation and the rare grace of an older and less hurried day than ours.

Dr. Goodell's name must remain for a long time prominent in the annals of gynecology. Of bold and enterprising intellect, he did much to aid the earlier progress of his branch of our art; and, later, his clear and forceful style and ardent mode of writing did as much to restrain the excesses into which the ease and safety of modern surgery have tempted too many of

the followers of the great minds to which we owe our earlier gains in gynecological treatment. Doubtless this Fellow's very interesting life will be the subject of elaborate reviews by some one of you more critically competent than I. I could not leave his name, however, without a word to express my personal sorrow for the loss of a constant friend, and my regret that a life so continually useful should have been cut short just when it seemed to have won the right to repose amidst the pleasant and well-earned surroundings of honorable success.

Our custom does not, as a rule, insist upon a memoir of those Associate Fellows who fall out of our ranks; but the name of Oliver Wendell Holmes represents and illustrates so much that is rare and valuable among us that you will, I am sure, pardon me if I say of him a few words as to the exceptional position he held in medicine and literature. I say in medicine and literature, for although medicine has given poets to literature often enough, an example of a man eminent in any line of our work, and also continuously productive as poet, novelist, and essayist, can hardly be said to have existed until the long and brilliant career of Holmes. You know, as we all do, the remarkable medical essays which first brought him into notice; you are as well aware of his forty years of anatomical teaching. Neither in this nor in his medical essays did he fall below the higher standards of attainment. Yet, during these many years he poured forth novels, essays, and, above all, poems in which wit, humor, and pathos were mingled as perhaps they never before had been in any product of the English tongue. But this is not the place to deal with the purely literary aspects of the life of one who went smiling at the world through his delightful verse for so many years. It is rather the combination in one career of poetry and the practical teaching of anatomy which strikes us as exceptional, and asks attention here. Nor should we omit to observe that to write well on medicine, to teach anatomy acceptably, are thus shown not to be incompatible with the large production of literature which has won the ear of the world. In fact, Holmes never ceased to be a physician in some sense, and to act and

think with and for us. He said to me more than once that he was sorry he had ever ceased to practise medicine. When he thought there was some risk that I might be seduced from the path of scientific medicine he warned me of the danger, and in so doing expressed on paper the regret I have just mentioned. Even as it was, he did us much good service, and never lost the chance with tongue or pen to exalt the profession of which he was proud.

You possess, I believe, the only remarkable portrait of this delightful scholar and physician. It hangs in your College Hall, where it will help his gay verse to keep him in the remembrance of those who did not know him in the flesh. I, personally, regret in him the helpful friend of nearly forty years. His good sense, keen logic, and stinging wit were ever at our disposal, whether for a laugh at our own follies or excesses, or a joyous raid of laughter-laden logic upon organized quackery. He did something, also, to emancipate us from narrowing social rules; he made it more easy for you and me to be independently what we will *outside* of medicine, if only *within* it we continue to prove our competency.

I have had it much on my mind that the College should continue to keep active touch of all public affairs which affect the health of the community, and should be ready to assist with advice or influence.

Some effort should be made in this State to protect by statute professional confidences. I submit to you for reference to Council a draft of the laws of other States regulative of this matter.

Again I remind the Fellows that the College can only hope for endowments through their own efforts with those outside of a body by no means able of itself to create such reserve funds. During my own terms of office the Library has thus obtained several small endowments.

I desire, also, to remind you that we shall be obliged, in the interest of our valuable book property, to use electric lighting, and to change our mode of warming to one less productive of dust.

You will not, I think, consider it out of place if I suggest that papers submitted to the College should not be overloaded with long details of cases, and that all communications should be limited to thirty minutes, a period quite long enough to announce novel views or discoveries, or for criticism of received practice.

I cannot take final leave of you without some expression of the feeling with which I give up the high office with which you have so long honored me. To be the presiding officer of the oldest and most renowned of all of our societies, with its long line of distinguished men, and having a history coeval with that of the Constitution of our country—this I look upon as the highest reward the profession can offer.

I thank you for the trust and for the confidence which that trust implies. That your affairs have greatly flourished during my terms of office is due to many causes, and chiefly to the efficiency which has always characterized your committees. I, myself, have left nothing undone which, in my opinion, could increase your prosperity or help to sustain the usefulness and the dignity of the College. I shall resign my place with the satisfactory feeling that you will replace me with one whose high reputation as physician and scholar will assure to you a firm rule, active helpfulness, and a sympathetic regard for those traditional methods of debate and social conduct which have been so rarely disturbed in my long acquaintance with your deliberations.

I ask that for a time you will not invite me to serve you officially in any way. Finally, let me thank you for the constant courtesy and kindness with which I have been treated for these many years by the Fellows. That is more than the gift of office, more than any office; something to take with me and to hold always in remembrance.

DISCUSSION

ON THE

ADVISABILITY OF THE REGISTRATION OF TUBERCULOSIS.

[Special Meeting held January 12, 1894.]

The Council to which the resolution and amendment with reference to the proposed action of the Board of Health concerning tuberculosis were referred, offered the following resolution to the College:

Resolved, That the College of Physicians believes that the attempt to register consumptives and to treat them as the subjects of contagious disease would be adding hardship to the lives of these unfortunates, stamping them as the outcasts of society. In view of the chronic character of the malady, it could not lead to any measures of real value not otherwise attainable.

That strict attention on the part of physicians in charge of the individual cases, insisting on the disinfection of the sputum and of the rooms, on adequate ventilation, and on the separation of the sick from the well as far as possible, will meet the requirements of the situation so far as they practically can be met, and better than any rules that, for diseases so chronic, can be carried out by a Board of Health.

That the College of Physicians respectfully requests that no official action be taken in the matter by the Board of Health, except the insisting on the disinfection of rooms in which consumptives have lived and died, in instances in which such procedure is not likely to have been adopted under the direction of the attending physician.

DISCUSSION.

Dr. Owen J. Wister: When I offered my resolution at the December meeting of the College it was objected by those who are in favor of registration of cases of consumption that nothing was intended beyond registration, in order that medical literature of a kind to warn persons of danger might be sent to their houses. In a paper read before the Pan-American Medical Congress on "Tuberculosis," and published in the *Medical News* of October 21, 1893, there is a good deal more in reserve which is insisted upon.

Registration, in the first place, is meant to include the residences of all persons affected with pulmonary tuberculosis, and even if the place of residence is temporarily changed it must be by and with the consent and knowledge of the Board of Health.

As to the literature, so called, I think that is of doubtful desirability. Those who spent the summer of 1892 in town, when there was a cholera scare, will remember that there was a great deal of this sort of literature—the papers reeked with it. The evening papers published articles of so sensational a character that the Health Officer was lashed into such a state of hysterics as to propose a quarantine against New York, under the act of Legislature which makes such a proceeding lawful when a contagious disease is "raging in a neighboring city." The "rage" in this case amounted to four cases in a month, in a population of a million. This proposition, however, was not adopted. But, while the Board of Health was thrown into spasms of terror, there was enough that was ludicrous in this literature to throw the community into spasms of laughter, and we had a rather cheerful summer. It cannot be hoped that this view will always be taken when, instead of a scare of two or three weeks, it will last ten or fifteen years, while from forty to fifty are dying a week. It is then possible that this literature may lash the whole community into panic, and that instead of regarding the unfortunate victims of consumption as objects of compassion they will be looked upon as peripatetic fountains of danger, and a feeling of hostility to them will arise. This may last for years, for during the whole period of softening they are regarded as sources of danger. In fact, they are to be treated as criminals guilty of consumption. As I said before, their residences, however temporary, are to be disinfected and their miserable lives are to be rendered more wretched by being haunted by the familiars of the Inquisition. It is asserted that if these radical measures are carried out tuberculosis will be eradicated in a few years; of that, it is said, there is no doubt. Now no other contagious disease has ever been extinguished, even with the added protection of vaccination in smallpox. Those of us who are not in the intimate confidence of Nature find it difficult to understand how an hereditary disease can be eradicated by measures which only limit its

spread by contagion; and if it is not hereditary, whence come those forms of tubereulosis other than pulmonary? It cannot be pretended that babies, a year or two old, get tubercular menginitis by contagion, nor can white swelling, nor suppurating glands, nor the many other exhibitions of scrofula come from contagion. I think very few of us will concur in this sanguine expectation. There are several other suggestions: one is likely to be very popular; it is that all persons working in factories and the like who are affected with pulmonary tuberculosis, in the softening stage, shall be withdrawn and, when necessary, supported by pensions. In this country we have a large experience of pensions; pensions have bankrupted the treasury of the United States. It is easy to believe that people unwilling to work could get the necessary certificate from some twopenny doctor and be supported by the State. As to the more serious part of the matter, the treating of persons so unfortunate as to have pulmonary tuberculosis as criminals guilty of consumption, it is something so frightful that I implore the College to interpose its remonstrance against such an outrage on common sense and common humanity.

Dr. L. F. Flick: Together with other Fellows, I would like to bring up the entire subject for discussion, and, without wishing to find fault with Council in referring back the resolutions in amended form, I should like to offer a substitute, which will bring up both registration and special hospitals for the treatment of the consumptive poor. The substitute is as follows:

Whereas, Tubereulosis is now known to be a eontagious disease; and

Whereas, The methods by which the disease is conveyed from the sick to the well are now clearly understood; and

Whereas, It has been shown that the room which is occupied by a consumptive during the infectious period of the disease, and the furniture and the bedelothing which have been used by him, become infected and are liable to convey the disease to others who may occupy or use them subsequently; and

Whereas, Tuberculosis, owing to its long duration, cripples the bread-earning capacity of the family, when it occurs among the poor, to such an extent that the want and hardships which follow in its wake prepare the healthy members of the family for the disease; therefore be it

Resolved, That we recommend to the Board of Health of the City of Philadelphia the registration and disinfection of houses which have been infected by tubereulosis;

Resolved, That we recommend to the City Councils of the City of Philadelphia the establisment of a Municipal Hospital for the treatment of persons suffering from tuberculosis.

The question of contagion seems to be admitted, and can, therefore, be eliminated from the discussion. I will take up the question of prevention. In reply to what has been said by Dr. Wister, I may state that the paper

from which he quoted was a scientific paper covering the entire subject. He ought to have told you that the author pleads in that paper only for a beginning in preventive measures.

The contagion of tuberculosis being admitted, what measures are necessary for its prevention? The careful investigation of this subject from many points of view by Dr. Cornet, Dr. DeForest, and myself, show beyond doubt that there is a local infection; that the consumptive in his home is bound in his ordinary daily life to infect his room, the furniture, and the bedclothing. The experiments made by Cornet illustrating this subject are most elaborate. They covered a period of two years, during which time he perseveringly followed out his line of research. He examined the dust gathered from the floor, the walls of the room, the bedposts, and the bedclothing of persons in advanced stages of consumption confined to their beds. He found by inoculation with this dust that he was able to produce tuberculosis. He found, moreover, that where a patient was sufficiently intelligent and obedient to follow the instructions to always spit in the sputum-cup and never into a handkerchief or on the floor, inoculation with the dust produced no effect; but where the patient occasionally spat into a handkerchief, or into a nook in the room, inoculation with dust from the surroundings of these places was effective. He was able to demonstrate that in proportion to the obedience and cleanliness of the patient the limits of the infected environment decreased. Where the patient was cleanly and took ordinary but not absolute precautions, the infection extended to only a limited space, possibly four or five feet. Where the patient was uncleanly and spat indiscriminately, not only the bedposts and the floor but also the walls were capable of conveying the infection. The clinical investigations which I made in this city, some illustrations of which I shall throw upon the screen, and the clinical investigations made by Dr. DeForest, in New Haven, corroborate these results in such a way that their meaning cannot be misunderstood.

I wish here to refer briefly to the results which I obtained in my investigations in the Fifth Ward of this city, and I shall throw on the screen sections of a map. This ward contains 16,000 people and has 3500 houses. I have tabulated the deaths from tuberculosis during twenty-five years:

Slide No. 1 shows probably the least infected section of the ward. There were comparatively few deaths from consumption in this section. Where deaths are noted they occur in groups—that is, in adjoining houses.

Slide No. 2. In this section some of the houses have had a number of deaths, while the majority are free.

During the twenty-five years there were 1500 deaths from tuberculosis in the entire ward, and they occurred in 650 houses.

Slide No. 3. This shows a section of the city inhabited largely by the colored race. Here the houses that have had the disease have had it frequently recurring. In this small street there is a house in which there have been deaths in five different families in succession, all occurring within a

few years. I was recently called to visit a rabbi in this house, who had never lived in any other house in the city. He moved into the house in perfect health and in six months contracted the disease. This was the fifth family in succession that had moved in and contracted the disease. Many of the other houses tell the same tale.

Slide No. 4. This section tells the same story. The houses that are always infected have a large number of cases in succession. These cases have not been in the same family, but have frequently occurred in different families.

Slides Nos. 5, 6, 7, and 8 show the same state of things.

Slide No. 9 shows a map of New Haven, which was prepared by Dr. DeForest, showing the deaths during seventeen years. His conclusions are exactly the same as mine. He found one interesting fact which I was not able to find because I did not have the entire city. He found that the most populous districts are almost entirely free. In the First Ward, with thirty inhabitants to the acre, there were scarcely any deaths, while in one section which has a large mortality the population is only six to the acre. That proves conclusively that crowding does not entirely explain the death-rate.

Slide No. 10 gives a section of New Haven and shows the same grouping as by map. It shows the recurrence of the disease in the same way as in the Fifth Ward of this city.

Now this evidence of infection and retained infection of houses, together with the demonstrations made by Cornet, can point to only one conclusion, and that is that the houses which have been inhabited by a consumptive for a given period will retain the infection and convey it to others. I have investigated quite a number of cases which illustrate the same point. I have knowledge of a telegraph office down in Maryland in which five consecutive operators contracted tuberculosis and died. The results in these five cases were so striking that people became superstitious in regard to the office. I have been informed by a person who saw the office that the consumptives had spat around the office and that it was lined with tubercular sputum. I have been able in this city to trace a great many cases where persons innocently moved into houses unsuspicious of any infection and where within six months a member of the family who was healthy before has taken the disease and this case has been followed by others.

Dr. DeForest mentions a very striking result of his investigations in New Haven. He investigated the histories of 100 cases coming to the medical clinic of the city and found that 52 were living in infected houses. In 1888 I carefully examined the deaths that occurred in the Fifth Ward during that year. There were 83 *bona fide* deaths from consumption. Although out of the 3500 houses only 650 were infected, two-thirds of the cases occurred in these infected houses. Of the remaining third a large number had lived in infected houses and had changed their residences, so that apparently more than two-thirds of the cases in which death occurred in 1888 had contracted the disease while living in infected houses.

If the facts I have related are true, how is it possible to institute any practical preventive measures without registration? The education that is spoken of in the resolutions offered by Council will not accomplish much. Cornet has shown that even where the patient has carried out most of the instructions the immediate surroundings are infected. As you are well aware, among the very poor there is no nursing. The consumptive is apt to lie on a bench in the kitchen—a small room, perhaps eight by twelve feet. He has to wait on himself and probably spits all over. Under such circumstances he is bound to infect the room, and it has been shown that the disinfection of such a room does not consist in burning sulphur, but special methods are required. One of the most efficient methods is rubbing down the walls with dry bread and then washing with carbolic acid or some other powerful germicide. The furniture and the bedding also require disinfection. You well know that the houses in which the poor live are owned by men who are trying to get as much money as possible out of them. The owners will not disinfect these houses if they can avoid it. It is only by authority of the government that this disinfection can be accomplished.

Now, is the profession ready to step forward and say "We will make an earnest effort to stamp out tuberculosis?" The profession throughout the country has said that it will. The American Public Health Association, during its session in Chicago, adopted resolutions asking that registration should be practised. The Section on Hygiene of the Pan-American Medical Congress adopted similar resolutions. The Congress of Tuberculosis, in Paris, in 1888, passed resolutions asking that tuberculosis be recognized as a contagious disease, and in 1891 passed a resolution asking that disinfection be practised.

Will disinfection have any effect? It will. I should like to cite one illustration: As far as I know, Berlin is the only city that has taken scientific precautions against tuberculosis. The result is most encouraging. In the city of Berlin there has been a marked reduction in tuberculosis from 1884 to 1891, the most rapid reduction of any city of which I can obtain statistics. In Philadelphia there has been a reduction. In London there has been a vast reduction since the establishment of consumptive hospitals fifty years ago. In Berlin, from 1884 to 1891, there has been a reduction of 0.644 per 1000. In 1884 the mortality in Berlin was 3.45 per 1000, and in 1891 2.811 per 1000. In Philadelphia, where we have been priding ourselves on the reduction, there has been in ten years only a reduction of 0.623 per 1000. Whether or not registration is being enforced in Berlin I do not know. I have been told by private parties that it is. Contrasting the reduction in Berlin with the reports from other cities, we find that the mortality in Paris was about the same in 1890 as thirty years prior—4.574 per 1000. Whether or not France will succeed in establishing preventive measures is yet to be known. I have had no definite information on the subject.

As to the sentiment on this question, it is all on the side of registration.

I grant that it may be hard for wealthy people to be recorded as suffering from tuberculosis, but this is a disease of the poor; the vast majority of consumptives are very poor, and the necessities of the poor so demand registration that it should outweigh the sensitiveness of the rich. The sensitiveness of anyone, however, can be overcome by the suggestion embodied in the resolution I offered—a suggestion which was made to me by Dr. James C. Wilson—that is, instead of registering the individual to register the infected house. It would then not be necessary to register tuberculosis among the wealthy, as only houses that had been infected would have to be registered. Where infection can be prevented by proper sanitary measures there would be no occasion for registration. This would enable us to register those houses that need to be disinfected.

The best place to study this subject is among the poor. It is there that I have learned my lessons. I am satisfied that every man here will reach my conclusions if he will go among the very poor and study this question at the bedside, and see how, one by one, these people die from the disease because they are unable to protect themselves and because in the endeavor to protect and supply medicine to the stricken one the healthy become victims. It is these that need aid. The College of Physicians should now take a firm stand and do something in aid of these people. It is not necessary to do everything at once. Let us make a beginning. Experiments have been made, let us now come to action. It is not fair to the public that the medical profession, with the knowledge it possesses, should do nothing. The laity cannot act because they have not the knowledge. If we who have the knowledge do nothing we are certainly responsible for the deaths of those who otherwise might be saved.

Dr. J. M. Da Costa: Let me say, in the first place, that I thoroughly admire the enthusiasm which Dr. Flick has for years brought to the study of this question. I think that the way in which he has gone about the work is in every respect most commendable, and in its spirit and scope this is one of the best series of observations with which I am acquainted. If I differ with him it is rather in the conclusions than with reference to his mode of investigation and his aims.

When we speak of contagion in consumption, we cannot mean that it is markedly contagious. There is no proof that it is. It is not contagious like typhus fever, or smallpox, or scarlet fever, or diphtheria. It is only slightly contagious. It is, indeed, so slightly contagious that some of our best thinkers with the largest fields of observation, notwithstanding the evidence that has been adduced, still hold that it is not contagious at all. For instance, if we take the opinion of a man so long connected with the Brompton Hospital as Dr. Williams, we find, in the last edition of his work on *Pulmonary Consumption*, that he maintains that no more deaths from consumption occurred in the attendants of the Brompton Hospital than occurred among the same number in ordinary life. The evidence of Dr. Andrew, of the Victoria Park

Hospital, is in the same direction. When we look at the conclusions of these men and at those of Wilson Fox and many others, it is going too far to assume that the whole profession admits the disease to be contagious.

That it is moderately so, and that it can be communicated under exceptional circumstances, I firmly believe; but that we should regard it as a very contagious disease and take all the precautions that we do in such diseases, I deny. If it be contagious, it is so, as every one admits, chiefly through the sputum. Is it to be understood that an inspector from the Board of Health is to come daily to take care of the sputum cup? Is not the intelligent physician the proper health officer? Is not what he says sufficient? Can he not advise and enforce the disinfection or the destruction of the sputum as well as any public officer? Finding bacilli in the bedclothes and on the bedstead, and destroying them, will not eradicate consumption. Tubercle bacilli are widely diffused. They are in the dust of the air we breathe, blown about from the dried sputum of consumptives in the street. They have been demonstrated to exist on the fruit sold in our markets; they are in milk. They have been found in churches, in places of amusement. They may, indeed, be said to be everywhere, and not only in the homes of consumptives. Where will you draw the line as to watching and interfering with the life of the consumptive? Must we not take the broader view, and act on the degree of the communicability and on the practicability of controlling it, rather than on the mere abstract question whether tuberculosis be contagious or not? The degree, every observer knows, is extremely slight.

The excellent series of observations about the houses is, I believe, subject to several fallacies. In the first place, unless you know the history of the persons themselves, these observations on the houses supposed to be infected are not conclusive. You must know whether or not the individual previously had or inherited tuberculosis. I am one of those who believe that the disease is largely hereditary. Of what use is it to say how many living in these houses die of consumption unless you know the history as well as the number of those who have occupied them?

Again, we should bear in mind when discussing the contagiousness of phthisis, how enormously prevalent the disease is, and how difficult it is to draw conclusions when you have the most prevalent chronic disease to deal with. As bearing upon the question of infection from husband to wife, it has been calculated by Longstaff as a mere matter of statistics without reference to the question of contagion, that of every 148,121 men who die of consumption in the ages of married life, there would be 4358 wives who would have consumption—that is, about 1 in 33. Thus, for every thirty-three married men who die of consumption there would be one woman have it, as a mere matter of ordinary frequency, whether the disease be contagious or not, and the probability would be a little more for phthisical wives. The number of cases seen in which both husband and wife have been affected is compar-

atively small. Flint, in over six hundred and seventy case, noted but five such examples. My record shows more such instances, including one where a tubercular husband had three tubercular wives. Still I have not met with very many, and the chances are always slight that the husband communicates the disease to the wife, or the wife to the husband. When we take all these facts into account, as well as the strong hereditary tendency of the malady, we must be careful how we draw conclusions in individual instances of apparent contagion.

Mindful of the observations of Dr. Flick, I have for some time questioned myself with reference to houses that I have known for years in which there has been consumption. But I have not taken them in one district, but everywhere. And this seems to me a much fairer way of studying the question, as the indiscriminate selection obviates the likelihood of error from bad hygienic conditions, especially of drainage, that houses near each other might share.

I have many houses in mind in which no case of consumption followed the first. In one, the father died of a slow consumption twenty years ago. The mother is, as regards tubercle, perfectly healthy to this day. The seven children have grown up into exceptionally healthy young men and women. In another house the wife died of consumption about eighteen years ago. The husband continued to live in the same house, and a family of children have grown up healthy. Another house has been occupied for many years by a consumptive who has had the disease for twenty-five years. Neither his wife nor any of the five children has become affected, though he is in the hands of a physician who does not believe in the contagiousness of the disease, and does not direct the sputum to be disinfected. Another house was occupied for years by a consumptive mother, whose husband had died of the disease many years before in another city. The son and daughter remained in the house mentioned for eight years in perfect health. It has since been occupied by a lady whose history I know, who is also healthy. The son got married about seven years ago, and has lived in various places. Within the last two years he has become a consumptive. His strong hereditary tendency determined it. I could go on citing instance after instance. I admit they furnish negative evidence. But negative evidence in a matter of this kind is valuable. I am on the whole quite certain that in by far the larger number of cases where I have known the history of both the house and of the household for a long time, there could not even be any suspicion of house infection. It is true also that in most of the houses cleanliness and ventilation were well attended to.

Further, in the question of house infection, we must not overlook the fact that these supposed centres of infection may adjoin, and have common drainage. And, under any circumstances, does it not suggest that possibly there is something wrong in the drainage or subsoil as much as it does infection of the house? The well-known observations of Bowditch, and of

others, have made us familiar with this mode by which consumption spread in Massachusetts and in the whole of New England.

With reference to hospitals: Consumptive hospitals have been brought forward as strong evidence of the non-contagiousness of phthisis, though I cannot say that the figures adduced are to me absolutely convincing, and there may be something in the greater prevalence and concentration of the poison that makes consumptive hospitals more likely to be sources of infection. I have already referred to the observations and opinions of Dr. Williams and of Dr. Andrew. But as regards general hospitals in which consumptives are, they certainly cannot be shown to be places of infection. I will quote the remarkable results in the General Hospital of Vienna. That hospital is one of the largest in the world. It is full of consumption, and there were, as we know by some observations made long since, and before the disinfection of the sputum was attended to, in three years 2736 deaths from phthisis, and not a medical officer or a nurse had become infected. I will refer to the record of the Pennsylvania Hospital, which always has consumptives in its wards. I have taken some pains to ascertain the truth in this matter. In my long connection with the institution there never has been a time when there were not cases of tuberculosis in the medical wards, and there have also been cases of surgical tuberculosis in the surgical wards. Of 147 resident physicians that have been in the hospital in the last seventy years, and of whom I have traced the medical history, and many of whom I have personally known and examined, but one has died of tubercular disease. Two others have at different times shown tubercular symptoms. One of these is now living in Arizona in fair health. In the other, even bacilli have disappeared from the sputum. The one dying, did not die for over five years after leaving the hospital. Moreover, before becoming a resident physician, he was in delicate health, so that it is not fair to attribute his death to his having been in the hospital.

A stronger statement still can be made with reference to the nurses. Of forty male nurses that have been in the hospital in the last twenty-five years, but one has shown any sign of tubercular disease. He is now a patient in the hospital. He did not become tubercular until four years after leaving. There have been 163 female nurses whose histories can be fairly well traced. Of 53 that have left the hospital in the last five years we know their present condition accurately. Not a single one of these has had certainly any tubercular symptoms, notwithstanding that there was always tuberculosis in the wards, always some exposure, and until recent years disinfection of the sputum was not practised, although ventilation was always attended to. There is only one case even doubtful. She has an occasional cough, and is not strong. Her father and mother both died of consumption.

When we take all these facts into account, granting, as I do, that up to a certain extent tuberculosis is contagious, I think that the recommendation of the Council to the Board of Health is the right one. Why fix the brand of

leper on a poor unfortunate because he has consumption, when the medical officer can do all that is necessary? He can instruct how to ventilate and keep the house pure, and how to disinfect the sputum. What more could a Board of Health do? What can it do, except in instances of death where proper disinfection may not be carried out? Where death has occurred, and where disinfection is not likely to be practised, it would be proper for the Board of Health to interfere. Under other circumstances, why should we place a stigma on the consumptive, why have him pursued from house to house, why have him a marked man, why have the house a marked one? Why give it a bad name and injure the landlord because there has been a death from consumption in it? I think that the resolutions offered by the Council cover the whole case. No human being will suffer. It will only enforce on medical men the necessity of insisting on proper disinfection and proper hygiene. It will do as much as possibly can be done, as much as any Board of Health can do.

With reference to hospitals for the consumptive, there is a great deal to be said on both sides of the question. Undoubtedly larger accommodations and better means of separating the sick from the well are desirable. But whether this can be made in any way obligatory is doubtful; large social questions and questions of finance arise which take the matter far beyond our power materially to influence.

We must not overlook the strong hereditary tendency to the disease. If you want ever to get rid of consumption, it is not going to be simply by the disinfection of sputum and similar means. It will be largely by the prevention of the marriage of tuberculous persons. There you strike at the root of the evil. Until hygiene, preventive medicine, and law have reached that point, I think that we are constrained to let this question take care of itself, doing the best we can to limit the ravages of the disease. I admit that it would be most desirable if we could separate the consumptive from the well. If the State were rich enough to make colonies in climates in which consumption will not flourish, that would be a most admirable means.

Let me say that it is not because I do not believe that phthisis is communicable that I oppose the contemplated action of the Board of Health to declare the disease contagious, and to register consumptives, but because I believe that the means proposed will produce hardship without corresponding value, and that they are both unnecessary and insufficient. The Board of Health has already the right, and may well enforce it, to have any house that, whatever the cause, has a bad sanitary record, put in better order. Moreover, it can do much in destroying other sources of infection, such as from diseased meat, from the milk of tuberculous cows, in improving drainage, in favoring open spaces that air and sunlight may get into houses. But let it leave the care of the individual where it belongs—to the conscientious physician.

Dr. John B. Roberts: It seems to me that this question is almost

exactly the same as the one with which surgeons struggled ten years ago. I was one of the conservative ones who did not at first practise antisepsis, but employed half-way measures and saw all my cases suppurate. This is the point to which the question of tuberculosis has come.

I think that Dr. Da Costa's statement that there are a large number of cases all over the country, which he uses as evidence that the disease is not certainly infectious, is really an argument on the other side. It is so because there is infection. There are so many hereditary cases because infection of the parents has so weakened the tissues that a lowered resistance is transmitted to the children, and they cannot repel the action of the bacilli.

It is said that the doctor can do all that is necessary, but Dr. Da Costa has stated that he has known of cases where the physician would not take any precautions. That is a reason why the Board of Health should step in among the poor and see that the houses are clean. We see this in diphtheria. It is not a question of damage to landlords. It would be an advantage if everyone knew that every house was disinfected, either by the willing efforts of the people, or owners, or by the Board of Health. There seems to be no reason why there should not be a report to the Board of Health if it will act in a reasonable manner. There is no expectation that the Board of Health would take the same measures as in typhus or typhoid fever. It is only a few years since typhoid fever was placed on the contagious list. If the matter had been discussed publicly there would no doubt have been many men on both sides—some for, some against, doing so.

It might be well to mention what has been done in other places. In February, 1892, at a conference of the medical staff of the Manchester (England) Hospital, with the medical officers of health and others, it was unanimously resolved that it was desirable that certain cases of phthisis should be notified to the medical officer.

Dr. Hermann G. Biggs, the Chief Inspector of Pathology, Bacteriology, and Disinfection in the Health Department, has sent to the Health Board a long statement regarding the contagiousness of tuberculosis, accompanied by a number of recommendations. He says:

"First. Tuberculosis is a contagious disease and is distinctly preventable.

"Second. It is acquired by the direct transmission of the tubercle bacilli from the sick to the well, usually by means of the dried and pulverized sputum floating as dust in the air.

"Third. It can be largely prevented by simple and easily applied measures of cleanliness and disinfection."

His recommendations include:

"That there be systematically disseminated among the people by means of circulars, publications, etc., the knowledge that every tubercular person may be a source of actual danger to his associates, and his own chances of recovery diminished, if the discharges from the lungs are not immediately destroyed or rendered harmless.

"That all public institutions, such as asylums, homes, hospitals, dispensaries, etc., be required to transmit to the Board of Health the names and addresses of all persons suffering from pulmonary tubercuIosis.

"That all physicians practising their profession in the city be requested to notify this Board of all cases of pulmonary tuberculosis coming under their professional care."

The North London Hospital for Consumption issues directions to its out-patients and to its ward patients, recognizing the contagious nature of tubercular phthisis and suggesting precautions.

The Royal National Hospital for Consumption, Ventnor, has this to say:

"Patients are earnestly requested not to spit on the ground, floor, or fireplace, but to expectorate into the proper vessel. When this is not possible, the handkerchief should be used without fail; but in order that the expectoration may not become dry it has been arranged that a clean pocket-handkerchief shall be supplied to each patient daily, the soiled one being at the same time removed for the double purpose of disinfection and washing. *It is to be distinctly understood that spittoons shall always be used when possible, and that the handkerchief is only supplementary to prevent spitting upon the ground.*"

The Manchester Hospital for Consumption takes similar ground:

"All matter coughed up from the chest should either be spat in the fire or should be received into a vessel lined in such a way with a piece of paper that the paper and its contents may be lifted out and burnt.

"Rags that can be burnt should be used instead of pocket-handkerchiefs, and if a pocket-handkerchief be used it should be well boiled before the matter upon it has had time to become dry and powdery.

"N. B.—The Medical Officer of Health for Manchester undertakes to purify, *free of cost*, any house that may be notified to him by competent medical men."

Last year the Northwestern Branch of the Society of Medical Officers of Health drew up a memorandum on the subject, which has been pretty widely distributed in the North of England. In this memorandum the infectious nature of consumption is definitely laid down, and dried sputum in the form of dust is indicated as the vehicle of contagion.

The County Borough of Oldham, in England, gives similar "precautions against taking consumption."

The French Ligue Préventive contre la Phthisie Pulmonaire says:

"The most frequent and powerful source of infection lies in the expectoration of consumptive patients. Although almost harmless so long as they remain in the liquid state, the sputa become especially dangerous when they are reduced to dust. They quickly take on this form when they are projected on to the ground, the floor, the walls; when they soil clothing, counterpanes, bedclothing, curtains, etc.; when they are received into handkerchiefs, napkins, etc."

With this evidence of scientific work in other places, can the College of Physicians of Philadelphia deny the contagiousness of phthisis, or doubt the advisability of the Board of Health knowing and registering the houses of at least the poor and careless, in which the germs of tubercular consumption are threatening the public safety?

While there may be a few who believe in the non contagiousness of consumption, the vast majority does believe that it is contagious to a certain extent. It is the same old story of antisepsis and sepsis. The College of Physicians should go on record that consumption is contagious more strongly than appears in the report of Council. In the second place, something should be done. That, it seems to me, should be in the direction of letting some official know that the consumption bacilli are to be found in a certain locality. Not the physician, but some central body should have the authority to ascertain the facts and enforce such measures as will limit the spread of the disease among the poor. If we limit it among the poor, we limit it among the rich.

DR. WILLIAM OSLER (Baltimore): The question may be thus briefly stated:

First. Following a primary law of parasitism, the bacillus tuberculosis frequents chiefly that organ in its host which communicates most freely with the exterior. Just as countless thousands of ova are thrown off from the intestine of the bearer of a tapeworm, so from tuberculous lungs in a state of softening and cavity formation, countless millions of bacilli are cast out daily with the sputa.

Second. The widespread diffusion of the parasite outside of the body has been demonstrated in the infectiveness of the dust and of the scrapings of the walls of rooms and wards occupied by patients with pulmonary consumption. Moreover, the greater prevalence of tuberculosis in crowded communities, the enormous mortality from the disease in prisons and institutions, and its frequent occurrence as a house malady, suggest that the conditions favoring its continuance are those which foster the growth and spread of a specific contagion.

Third. In the language of parasitology the lungs constitute the chief seat of election! But, apart also from gross pulmonary lesions, the proportion of autopsies in which the bronchial glands are found tuberculous, speaks unmistakably for direct infection in the exercise of their function as dust filters.

On these grounds I believe that the registration of pulmonary tuberculosis would be beneficial—enforcing attention to those sanitary details so apt to be slighted or overlooked, and diminishing directly the danger of contagion in the community.

Infection through food is closely related also to the endemic prevalence of tuberculosis. The incidence of the disease in the mesenteric glands of infants indicates that the gastro-intestinal canal is a portal of infection only a little less wide than that of the respiratory system.

The question of tuberculosis is not, however, settled with the disinfection of the sputa of consumptive patients.

The hereditary transmission of the disease must be accepted, though an estimate of the frequency of this mode of infection must necessarily be uncertain, but for certain forms Baumgarten's theory of latency is particularly suggestive. Tuberculosis has been well called the *pébrine* of the human race. The analogy is striking, for not only, as you will remember, is the parasite of the silkworm disease transmitted by direct contagion, but it also infects the eggs, which hatch, and may pass through various stages of development before they are finally destroyed.

Lastly, and here is consolation: the conditions which render individuals more or less immune scarcely yield in importance to those which maintain the vitality of the tubercle bacilli in a community. So widespread is the seed that few of us escape infection, and the statistics of the Paris Morgue show that in more than 50 per cent. of adults the germs not only gain an entrance, but actually effect a lodgment. As a factor in tuberculosis, the *soil*, then, has a value equal almost to that which relates to the *seed*, and in taking measures to limit the diffusion of the parasite let us not forget the importance to the possible host of combating inherited weakness, of removing acquired debility, and of maintaining the nutrition at a standard of aggressive activity.

Dr. H. C. Wood: I came here not to speak but to learn, and thus far have been quite successful. I wish to quote from a letter from Dr. Billings which has been placed in my hands. He says: "I should like very much to hear the discussion, but probably should have little to say, since I am doubtful as to what the decision of the College should be on this point. I presume that there are about six thousand people in Philadelphia affected with consumption, and that a considerable proportion of these have contracted the disease in infected houses. If it were possible by a systematic notification for the Board of Health to locate a considerable number of these infected houses, what steps would it take to purify them? How would it deal with those of the poorer classes who are affected with this disease, and with their furniture, bedding, and rooms? Until these questions are answered, I do not find it possible to form a definite opinion as to whether it is worth while to put in force a compulsory system of notification. Some of the worst infected rooms are probably those occupied by the criminal class, which is especially liable to this disease. For example, the Eastern Penitentiary is certainly thus infected; but in this I presume the Board has no jurisdiction.

"It occurs to me, that if the system of notification is to be tried, it might be best to commence with a limited area of the city in which the death-rate is now high; for example, the Seventeenth or the Seventh Ward, and see what results in diminishing the proportion of cases in this area can be obtained in the course of two or three years."

This thought has occurred to me, and has not been touched upon by any of the speakers: In a case of diphtheria the Board of Health is notified; the patient dies or gets well, and the room is disinfected. Suppose, however, a case of consumption: The Board of Health is notified, the house is disinfected to-day, but reinfection occurs to-morrow, and the end need not come for four or five years. Unless you add the power to forcibly remove the patient and shut him up in a hospital-jail where you can confine and permanently disinfect him, I do not see how you are going to get any good results from the process of disinfection. As far as disinfection goes, unless you can prevent to-morrow's reinfection, I see little use in to-day's disinfection. If, however, the Board of Health could disinfect houses in which phthisic cases have died, something might be achieved, but the ordinary death notice is all the notification required for this.

Dr. Thomas J. Mays: In the charts which are presented to us it is to be noted that the disease is less frequent in those parts of the Fifth Ward in the neighborhood of Spruce and Pine streets, and becomes more prevalent as we approach the poorer portion of the city. Dr. Flick's investigations extended over only a limited period of time, say twenty-five years. In that time he finds that about 25 per cent. of the houses in the Fifth Ward have become infected. I hold that if the investigations had extended over a longer period—say fifty, seventy-five, one hundred, or one hundred and twenty-five years—he would have been able to show that nearly every house in that ward was infected—according to the contagion theory, that in nearly every house in that ward a consumptive had lived and died. The same argument holds against the investigations made in New Haven. I have in my possession a book reporting similar investigations made by Dr. Riffel in two old towns along the Rhine in Germany. He investigated the death-rate from phthisis in these towns for fifty, seventy-five, one hundred, and in some cases one hundred and fifty years back. He found that in one town at least —Karlsdorf—at some time or other nearly every house had had a case of phthisis in it. I think the same thing would hold in regard to the Fifth Ward of this city if the statistics extended back sufficiently long; but as these only cover a period of about twenty-five or thirty years this is out of the question.

Dr. Flick also refers to the diminution of the phthisis mortality in Berlin, London, and Philadelphia. I think that if he had consulted the death-rate from phthisis in these cities he would have found a gradual diminution in the death-rate before the bacillus era began in 1882. I went over this subject some years ago, and found that in every large city there was a gradual diminution in the death-rate. Dr. Flick claims that the diminution in the death-rate of consumption, since 1882, in this city has been due to the disinfection of the sputum. His own figures show, however, that there occurred a larger number of deaths from this disease during the nine years following 1882 than during the nine preceding years. In what

way this makes a favorable showing for the theory of disinfection I am at a loss to understand. So far as I know there has been no Board of Health regulation in this city, nor in Berlin, nor in London, nor in any other city, yet their deaths are gradually diminishing in number. If the gentlemen on the other side will look backward a hundred years they will find that the very experiment which they are so anxious to try now was tested with the utmost rigor, from 1782 to about 1855, in Naples. It was decreed that every physician who neglected to report a case should be fined one hundred and eighty dollars; that the ceilings, walls, floors, doors, and windows of the room in which consumptives died be torn out and burned; that the bedding and furniture be also burned, and that such dwellings were not to be occupied for a year. The result was that the family with phthisis was shunned and driven to want. Houses in which consumptives died depreciated in value. The sick were neglected and left to die inhumanly away from their families and friends. Did any good result from these laws? Brehmer states: "Concerning a diminution in the rate from phthisis in Naples the medical historians of that period are ignorant." According to Uffelmann (*Berlin. klin. Wochenschrift*, 1883, p. 369), Dr. de Renzi, the medical historian of Italy, states that the injury which had been inflicted on Naples by these laws was simply indescribable, and denounces the Neapolitan faculty in the severest terms for participating in their introduction.

In vol. xlv., p. 112, of the *British and Foreign Medico-Chirurgical Review* it is stated that Drs. Spatuzzi and Somma, "who have paid great attention to the mortuary returns in that city (Naples, about 1860), affirm that a sixth or a seventh of the whole mortality is due to phthisis," and that Dr. de Renzi "marvels greatly (in 1863) that the city of Naples is fully as much liable to phthisis as either London or Paris, though the salutary condition of the climate should render it far less common."

If the death-rate from consumption was the same in Naples at the time segregation ceased as it was in other cities in which segregation had not been practised, it is self-evident that such a measure can have no influence in diminishing the death-rate from this disease.

Dr. James Tyson: I had intended to say something on this important subject, but almost every point which occurred to me has been so well treated by others that I hesitate to add anything. I admit all that has been said with regard to the contagiousness of phthisis, as determined by experiment, but as to the actual spread of consumption from one person to another as the result of association—as to the degree of contagiousness, in a word—I agree entirely with Dr. Da Costa. Admitting the correctness of Dr. Flick's studies and Cornet's observations, it still comes to this: that the physician is competent to take care of this question without the intervention of the Board of Health. Just as the surgeon is qualified to take care of the matter of antisepsis and asepsis in his management of surgical cases, so the physician is competent to take care of the disinfection of utensils used by

consumptives and the destruction of the germs which may be the cause of the disease, the more especially as such disinfection is acknowledged to be of the easiest kind. What Dr. Roberts has just said with regard to the situation ten years ago as to sepsis and antisepsis, could as well be alleged in favor of the position that I take—that the physician is competent to handle the difficulties. The surgeon did not call in the Board of Health to help him under like circumstances, but fought the battle alone, and won it triumphantly. The question has been studied experimentally and chemically, and we know exactly where to put our fingers to check the spread of consumption so far as this is possible. We must attack the disease in two ways: first, the contagion must be destroyed as far as possible with the means at our disposal, and second, we must seek to change the soil on which it is known that the bacillus flourishes, by improving the general health of the individual, and thus increase his ability to repel the invader. I feel that we are entirely competent to handle this question without the aid of the Board of Health.

Dr. Abbott: I have listened with much interest to both sides of the discussion, but must confess that I am not yet quite secure in my mind as to the proper course to be pursued. I am strongly inclined to the views expressed by Dr. Billings in his letter to the President. Tuberculosis is a widespread disease with manifold expressions, and if our efforts are to be directed against it, in the full sense of the term, the problem is certainly beset with many difficulties.

In connection with the subject under discussion, the question that has constantly presented itself to me is: If the College sees proper to recommend that all houses in which the disease is located be reported to the authorities, has it any guarantee that their condition will be improved? The object in reporting these houses is to secure complete disinfection of them, a process requiring conscientious attention to most minute details, to say nothing of a full knowledge of the requirements of the cases. If the Board of Health is in a position to assure us that these demands can and will be met, then I am in favor of the resolution. If not, then we have had a long discussion with but little result.

The ultimate object aimed at in the resolution I consider as most desirable, but will it be attained if the resolution is passed? We have abundant evidence in justification of the belief that localities occupied by persons suffering from the pulmonary form of tuberculosis, particularly, may and do become centres of infection, and it is highly desirable that such places should receive the attention that their character demands. Much has been said here this evening concerning the prevention of the spread of this disease by disinfecting the sputum, and this point has been accentuated as if the danger ended here. The danger does not end here. Sputum collected in a receptacle may remain perfectly harmless without disinfection or any other treatment whatever. The danger lies in the sputum *not* collected, but spat upon the floor, dried, ground into dust by the feet of passers-by, and ultimately

inhaled into the lungs—a condition of affairs often seen in dwellings of the poor. The predominance of pulmonary tuberculosis over other manifestations of the disease is certainly, in part, accounted for in this way. Dr. Da Costa in his remarks has said that "bacilli" are everywhere present, and if this is the case how are we to escape them, etc.? His use of the term bacilli in its generic sense is misleading; I can hardly think he meant that *tubercle* bacilli were everywhere present, for this is not the case. They *may be* present everywhere that tuberculous individuals are present or have been present, but to say that they *are* present in all places does not accord with the results of observation.

Dr. Frank Woodbury: The facts collected with so much commendable diligence by Dr. Flick are susceptible of quite another interpretation. They certainly do not prove consumption to be contagious disease, or that the bacillus tuberculosis is its sole cause. If he had confined himself to the task of showing that want of sunlight, dampness, and other mal-hygienic conditions were efficient causes of phthisis his data would have been equally applicable. If he had intended to prove that the habitations of some of the poorer inhabitants of this city were likely to act injuriously upon the occupants, so as to predispose them to phthisis, we might have been willing to go with him almost to any extent in making application to the Board of Health to destroy these sources of disease. We might admit that by adopting such drastic measures the mortality from tuberculosis would undoubtedly be greatly reduced. The total extinction of pulmonary consumption, under present conditions of civilized life, however, is entirely impracticable, and, to my mind, chimerical.

It may be admitted, if the proposed plan of registration and inspection were rigorously carried out, that the results would apparently support the claims of the author. The number of cases reported would certainly diminish to a remarkable degree; but the real explanation, however, of the diminution would be that a large number of cases would be concealed, and deaths would be reported as from other causes. Moreover, a large number of cases would object to the solicitude of the Board of Health for their welfare and leave the city. Physicians would have their sympathies appealed to by relatives, who might wish to conceal from the patient the fatal character of his malady, and would be in a dilemma between duty to their patients and duty to the State. In fact, registration would eventually divide physicians into two classes—those who reported their consumptive patients and those who did not. The physicians having the reputation of not reporting their cases would naturally have a larger *clientèle* than others.

The discussion of the registration of consumption manifestly brings up the question of the nature of phthisis. It is true that there are other clinical conditions which resemble tuberculosis of the lungs and which are not due to the presence of tubercle bacilli. Assuming, however, for the moment that all cases of consumption are tubercular, we may ask how large a part in disseminating the disease is played by contagion? We know that Dr. Brush

calls attention to an infected milk-supply as the principal source of tuberculosis in our cities. Bowditch showed conclusively that dampness of the soil is a very powerful factor in the causation of phthisis. Other authors claim that it is mainly due to inheritance. If these views may be regarded as well established, then infection sinks to a secondary place. If tuberculosis be the *pébrine* of the human race, as has been stated, this invites a consideration of the subject from the standpoint of comparative pathology. If phthisis be analogous to the fungous diseases of the lower animals and plants, then the results of treatment in the latter may throw light upon the therapeutics of human tuberculosis. In a communication made by Dr. George B. Wood, in 1869, to the American Philosophical Society he referred to a discovery which he had made, and which, I think, has a very important bearing upon this subject. He proved by experiments upon a large number of fruit trees, which were prematurely dying of progressive decay (now recognized as some form of fungous disease), that by simply adding potash to the soil the trees were restored to their early vigor, and they resumed fruit-bearing; in fact, the crops were larger than ever before, "many branches breaking down with their load of peaches." This is a striking illustration of the importance of supplying to the soil some chemical agent which may be deficient, rather than devoting our time and energies to the destruction of germs. Pulmonary consumption certainly is curable. How many have been cured solely by an antiseptic or a parasiticide treatment? Not one; but many have been restored to health by hygienic methods alone.

Let me briefly quote, without comment, a case in point. It is that of a distinguished Fellow of the College, the late Dr. Joseph Parrish. Dr. George B. Wood, who delivered the obituary address before this College, thus alludes to Dr. Parrish's illness: "When about twenty-five years of age the Doctor was affected with a severe and lasting cough, and considered himself in danger of pulmonary consumption, to which he believed that he had a family predisposition, having lost a brother and sister by that complaint. Under a course, however, of vigorous exercise, and exposure to the air, without the use of medicines, he ultimately surmounted the threatening symptoms. The existence of cicatrices in the upper portion of the lungs, discovered upon post-mortem examination, proves that his apprehensions were well founded, and at the same time affords strong evidence in favor of the plan of treatment which he adopted in his own case, and always strongly advocated."

Recent investigations into the etiology of tuberculosis seem to lead us away from the germ theory, and to point to the fact that in the tissues of the phthisical patient some chemical agent is deficient, which if we can supply, the symptoms will disappear and the case proceed to recovery. This agent may not be the same in different patients. Cases of consumption have been cured by the hypophosphite treatment advocated by Churchill; others have been restored to health by alcohol; yet others have been equally improved by drinking blood at the abattoirs, or by eating raw beef. Cod-liver

oil, advocated by Bennett, seems to supply the needed element in some; an abundant milk diet has been successful in others. The agent may be a simple salt like lime hypophosphite or sodium phosphate, or organic in its nature like tuberculocidin or nuclein. Whatever it may prove to be, one thing is certain, that the results from following out this lead have been more successful than those from parasiticide treatment. They do not strengthen the argument of those wishing to class tuberculosis among contagious diseases.

In conclusion, I submit for consideration that if the Board of Health should force physicians to make a diagnosis this would involve the compulsory use of all modern methods, including the microscope and culture-tubes. The fact has just been announced that ex-Vice-President Morton's fine herd of Jersey cattle, recently exhibited at the World's Fair, has been found to be infected with tuberculosis It is said that the animals presented no symptoms of disease whatever, and the diagnosis was made by the subcutaneous injection of tuberculin. Can physicians be compelled to resort to this method in all suspected cases in the human subject?

Dr. S. Solis-Cohen: The question to be considered is simply this: Granting all that may be said concerning the etiological relations of the bacillus of Koch to tuberculosis, and granting all that is said as to the spread of infection from persons affected with tuberculous disease: on the whole, will the health and comfort of the community, including both the well and the sick, be better promoted by having all cases of tuberculosis registered or by not having them registered? So far as the sick are concerned, the disadvantages of registration are obvious. So far as the well are concerned, my distinguished teacher, the Vice-President of the College, has shown that the reasons advanced in support of the proposition that it is necessary to have cases of tuberculosis registered in order to protect the health of the community, cannot withstand critical analysis. I believe, moreover, that registration would be a detriment to the community, for the reason that when of two measures to prevent an evil, attention is concentrated on the less efficacious, the more efficacious is likely to be neglected. If we direct our attention too strongly toward germicidal measures we shall lose sight of the more important measures that relate to the general sanitation of cities, of houses, and of individuals. Tuberculosis among men is to be prevented by increasing the vitality of the race, by preventing the marriage of tuberculous and otherwise unfit persons, and by perfecting the sanitary environment and life of the individual. The true prophylaxis against tuberculosis begins, therefore, before conception; but as society has not reached the stage of development where such prophylaxis can be generally instituted, it becomes all the more necessary to insist upon those practical measures of individual and civic hygiene which must be our greatest dependence, and to oppose whatever shall tend to obscure their importance.

As has been said by Dr. Woodbury, if Dr. Flick had started out to prove that consumption flourishes in overcrowded places and where misery, poverty,

filth, and vice are congregated, his statistics could not be more admirably adapted to enforce that conclusion; but to prove that tuberculosis is contracted only or chiefly by living in houses in which persons having tuberculosis have previously resided, would require that a census be taken to find out concerning every house in the city whether at any time there had been a patient with tuberculosis living or dying in the house, and if so, how many cases had followed and at what intervals, and also to determine how many cases of tuberculosis had developed among those living in houses where no one having tuberculosis had previously resided. Until statistics of this character are at hand, the case against the so-called infected houses, in districts where the city, the houses, and the lives of the people furnish so many potent causes of impaired vitality, is at least "not proven." But grant that the proof of infection in houses is conclusive. Suppose that the houses are thoroughly disinfected, and even, referring to Dr. Wood's point, guarded against reinfection—what have we gained? We are told—and I invite correction from Dr. Abbott, if I am in error—we are told by the highest authorities that the bacillus of tubercle is about us everywhere. It exists, save at certain altitudes, or upon the sea, or in other specially favored regions, in the air we daily, momently breathe. Everyone is at some time exposed to the danger of inhaling it, and that it is inhaled by everybody is shown by the results of autopsies upon persons having no tuberculosis elsewhere, but in whom the bronchial glands are infected with the tubercle bacillus. I believe that, according to some statistics, the proportion of persons so infected has been placed at 50 per cent. Is it not clear that, as was said by the President of the College in his Annual Address, in order to make the Board of Health laws effective, there would have to be instituted a system of espionage so that the tuberculous patient could be followed every day from the time of his rising up to that of his lying down, while seated in his house or while walking by the way, to make sure that by no possibility should his sputum be deposited into any place where it could become dry and be carried into the air—a thing which is manifestly impossible. But if instruction only is to be given and no such rigorous supervision of individuals attempted, it is within the province and within the power of the attending physician to instruct his patients, and the intervention of the municipal officer is not required.

Therefore, because of the impracticability of this registration to effect anything positively, and because, negatively, it will have the effect of diverting attention from the fact that these streets or alleys, to which Dr. Flick directs our attention, should be cleansed and widened, the houses properly drained and ventilated, the people properly fed and clothed, and otherwise protected from exhausting and depressing influences, I consider it to be the duty of the College to place its strong word on record against the proposed legislation.

Were the proposition in favor of a municipal hospital for tuberculosis

separated from the other recommendation, and properly safeguarded, I should be glad to vote for it.

Dr. Richard A. Cleemann: One very important point has not been alluded to. Statistics show that the death-rate from the disease is almost the same in every community, year after year. If there is any diminution, it is very small. This is directly opposed to the idea of the contagiousness of the disease. All other contagious diseases vary very much in their death-rate at different times. There is only one explanation by which we can conceive that phthisis is contagious and the death-rate always the same, and that is, that the whole of the community is thoroughly infected with the tubercle bacillus, and that in the community there is a fixed number who are susceptible to the disease. Just as, statistically, we can calculate how many giants or how many dwarfs are in a community, so can we estimate the number of persons susceptible to phthisis.

In this view of the case, with the bacillus tuberculosis spread over the whole city, measures to be effective must be of a very extensive character. The mere reporting of a case now and then—for I believe the rule will be often neglected—will have very little effect. Measures to be effective must be thorough, and they are the methods of general hygiene.

Dr. James B. Walker: We must look at the practical side of this matter, and ask what will be the benefit from registration? How much has typhoid fever in this city been diminished by the fact that we report our cases? Not in the least. The disinfection of the stools which the physician has directed has lessened the number of cases, as similar attention to the sputum may in phthisis. I am against placing phthisis on the list of diseases to be reported, for many reasons. I do not believe that this would confer a single benefit upon the city or its inhabitants. The physician is capable of doing all that the Board of Health can, without the manifold evils and annoyances of public registration. The College should go on record against the registration of the disease. It should go on record also as believing that the disease is possibly contagious, and should recommend physicians to use the most strenuous efforts to prevent the transmission of the contagion.

Dr. Da Costa: I used the expression that the tubercle bacilli were everywhere. A bacillus that is on the pavements, and in houses, that is wafted about in the air, that exists in milk, and on fruit sold in the markets, and, as has recently been demonstrated, is on the very money that we handle, may well be described as a bacillus that is everywhere. The tubercle bacillus is so diffused that it can, indeed, be considered as everywhere present, and it is scarcely a figure of speech to so describe it.

With every word that Dr. Roberts has said, I agree. There is not a direction he has read I would not subscribe to and endeavor to enforce as an officer of an institution, or in private life. I hope, indeed, that every Fellow will go away to-night imbued with the necessity of carrying out all measures that

have been brought forward with the view of destroying this scourge. But is it not better that we should do this as individual health officers, than to have it done in a necessarily perfunctory manner by a Board of Health? The only way that the Board of Health could radically act to destroy the tuberculous material would be to burn every house that was supposed to be really infected, separate the tubercular husband from the wife, take every child away, and cause such an upheaval in society as no community would submit to.

Dr. Flick: Dr. Williams, of the Brompton Hospital, has been quoted as an opponent of the theory of contagion, but he has written a paper in which he shows that a large number of nurses of the Brompton Hospital have contracted tuberculosis. I think that he has traced some fifteen or twenty cases.[1]

As to the ubiquity of the tubercle bacillus, Koch and Cornet have disproved it. They have shown that the contagious environment of tuberculosis is limited. Cornet has made experiments with dust taken from the streets and many other places at random, and was unable to produce infection by inoculation. He has, moreover, made an investigation of the health of the street-cleaners of Berlin, and has found them freer from tuberculosis than any other class. It must be borne in mind that the isolated cases of infection of fruit and other articles which have been referred to are traceable to handling by consumptives. Many of the fruit-stand dealers have consumption.

With regard to the endeavor to ascribe the occurrence of the disease in certain houses to dampness, that can be disposed of in a moment. If you will take one of the worst infected streets in the Fifth Ward, say Bay Street, you will find that at least 20 per cent. of the houses have not had a death from tuberculosis, although the sanitary condition is as bad as in the infected houses. Dampness can have no influence here.

As regards infection in hospitals, as I have said, Williams has written a paper in which he shows that fifteen to twenty nurses have contracted the disease in the Brompton Hospital. Cornet has written an elaborate history of hospital infection. He has taken the general hospitals of Germany in which the nurses are permanent, and he finds that as high as 62 per cent. had tuberculosis. In my own class of residents at Blockley, sixteen in number, two died within three years of the disease, and I myself was believed for a time to be a victim.

It has been argued that we ought to trace the history of these houses for a longer period, say one hundred years. There is a limit to the infecting power of tubercle contagion. What that limit is I cannot say; probably not very long. Unless cases occur within a few years of each other to keep up the chain of infection, the houses are no longer infected houses.

Italy has been quoted as an evidence of the non-effectiveness of preventive

[1] British Medical Journal, September 30, 1882.

measures. I am surprised that this quotation is made so frequently after what has been written. Those who have seen my paper on tuberculosis in Italy will be in a position to refute this statement. I have proved in that paper by careful quotations from contemporary authorities—and authorities that cannot be doubted—that at the time when the laws were instituted in 1782 the mortality was about 10 per 1000, and that when the preventive measures were abandoned, the mortality was so low in the extreme southern part of Italy that it was practically *nil*—0.19 per 1000. Instead of there being no result, the mortality was reduced from 10 to a fraction of 1 per 1000. Many who have written upon the subject have overlooked the fact that it is not Naples but the Kingdom of Naples that is spoken of. This comprised the southern half of Italy and Sicily. At first the northern part of Italy opposed the view of contagion and refused to accept any preventive measures. After some years it adopted preventive measures, but even at this day the mortality in the northern part of Italy is twice what it is in the southern part. These preventive efforts proved another point, and that is the local infection. The only practices that were of any benefit in the Kingdom of Naples were, first, isolation; and, second, compulsory disinfection. The preventive measures circled entirely about the household. The Italians did not know where the infection came from, but adopted crude measures of prevention. Yet with their crude measures they reduced the mortality to a minimum, and except for the fact that Italy became a health resort for tuberculosis, it probably now would have no tuberculosis. If you will study the statistics you will find that those parts of Italy that are health resorts for tubercular patients run up the mortality. This has been as high as 4 per 1000 in some of the resorts, but if you take the rest of the Kingdom of Naples, the mortality is a fraction above 1 per 1000.

As to the competency of the physician to meet this question. In connection with this subject, we must bear in mind that every infectious disease is a law to itself. It is not fair to apply to tuberculosis the laws which govern diphtheria, smallpox, etc. The infecting material in tuberculosis is easily controlled. It comes from a single source. In smallpox it is difficult to control. If it were possible to sterilize all the sputa of consumptives we would not need disinfection. Such a thing is impossible, however, and therefore it is necessary that the room be disinfected. This requires great care, and unless the family is willing to undergo the expense it cannot be accomplished. If the physician were to devote half of his time to bringing about proper disinfection he could not do it effectively.

Dr. Cleemann has advanced an argument against contagion, which, strange to say, has been made for the other side by Dr. De Forest. Dr. De Forest claims that inasmuch as the number of infected houses is limited, the mortality-rate should remain the same every year, because the number of exposures remain the same. The centres of infection remaining the same, the deaths should remain the same.

Tubercnlosis and typhoid fever have been compared. The reporting of typhoid fever cannot bring about any results unless the lessons taught are acted upon. If the water-supply were made better we should have no typhoid fever. There is no analogy between typhoid fever and tuberculosis. Tubercnlosis is infectious from the point at which it exists; typhoid fever infects our drinking-water. There is little danger from the room occupied by the typhoid-fever patient. The cases are not at all analogous.

Dr. Mays: When this question was discussed before the County Medical Society, about four months ago, I quoted Dr. Williams, the same authority to which Dr. Da Costa referred to-night in support of the non-contagiousness of the disease in the Brompton Hospital.

I read his conclusions from his book (*Pulmonary Consumption*, pp. 87–89) published in 1888, in which he emphatically states that contagion in the wards of this hospital is absolutely unknown.

In concluding the discussion of that evening, Dr. Flick said that in a private communication, which he had not with him but which he received from Dr. Williams since the book I quoted from was published, the latter admitted that his ideas had undergone a change in this matter, and that now he believed that about thirty or forty cases of phthisis had arisen in the Brompton Hospital through contagion, during the time over which his research extended. This seemed to me so extraordinarily strange and surprising—for, besides the conclusions set forth in Dr. Williams' book, I heard him say in one of his lectures at the Brompton, in 1883, that not a single clear case of contagion had occurred in the wards of that institution—that I determined to write to him and ask for the cause of this change of opinion on his part. I received his reply last October, in which he states that he has not changed his views on the infectiousness of phthisis materially since he summed them up in the second edition of his work on *Pulmonary Consumption*, pp. 87–89 (precisely that which I quoted), and that there are certainly no records to support the statement that thirty or forty cases of infection occurred in the wards of the Brompton Hospital. In fact, he says: "The evidence of the Brompton Hospital is all against infection, but we ought to admit that infection is possible through dried sputum under very exceptional circumstances, but that the slightest attention to cleanliness precludes any chance of this infection."

In this evening's discussion Dr. Flick again refers to this private evidence which he received from Dr. Williams, but he now states that instead of thirty or forty cases of phthisis which could be traced to infection in the Brompton Hospital during the time referred to there were only fifteen or twenty. It seems to me, therefore, that for the sake of scientific accuracy, and for the purpose of enlightenment, Dr. Flick ought not to withhold from us any longer the information which he claims to possess on these contradictory points.

Dr. Flick: The statements which I made are contained in an article by

Dr. Williams, which appeared in the *British Medical Journal*, September 30, 1882.

Dr. Charles W. Dulles: This is a discussion in which exactness is of great importance, and in which approximate figures and inferences must not be permitted to take the place of actual numbers and of facts. The danger of venturing on unfamiliar ground is exemplified in the comments on what Dr. Cleemann has said in regard to typhoid fever. Those who have specially investigated the sanitary condition of Philadelphia and the condition of its water-supply know that what Dr. Cleemann says on these subjects can be relied upon, for he has given to them unusual and unusually conscientious study.

Although when I came here I had no idea of speaking, I think it might be said for some who have so far kept silent that we do not to-night hear for the first time of Cornet, or of the Brompton Hospital and Dr. Williams, or of the Ventnor Hospital. The facts and theories emanating from these are all things about which we know something and about which we already have opinions.

In regard to the improbability of the effectiveness of measures of inspection instituted by the Board of Health, I agree with Dr. Walker. We have all probably had personal experience of the unsatisfactory way in which the Board of Health carries out its regulations in regard to diseases now returnable as contagious.

In passing upon the question before us I feel sure that the College will act in keeping with its traditions of a hundred years, and give a new illustration of the fact that it is a body in which prudent and conservative counsels prevail, and I have no fear that it will hastily commit itself, as a sister society has done, under the ingenious arguments of those strongly infected with the idea of the infectiousness of tuberculosis.

A vote was then taken on the amendment offered by Dr. Flick; it was lost, and the original resolutions, as proposed by the Council, adopted, the President being requested to appoint a committee of five to present them to the Board of Health.

HYDROPHOBIA IN THE UNITED STATES IN THE LAST FIVE YEARS; WITH SUGGESTIONS AS TO TREATMENT.

By CHARLES W. DULLES, M.D.

[Read February 7, 1894.]

The last time that I addressed a company of medical men on the subject of hydrophobia was in June, 1890, when I made some extemporaneous remarks upon the subject at Pittsburg; my last communication in regard to it which has been published is my Report on Hydrophobia, made to the Medical Society of the State of Pennsylvania, in June, 1888, which was printed in the volume of *Transactions* of the Society for that year. Since that time I have continued to collate material in regard to this disorder, and I now desire to lay before the College the results of a study of all the cases which have come to my notice since June 1, 1888—a period of five and a half years.

I have endeavored to get accounts of all the cases of so-called hydrophobia reported in the United States, and in doing so have availed myself of medical journals, daily newspapers of Philadelphia, New York, and Chicago, and a considerable correspondence. A number of cases, first heard of through telegraphic and necessarily brief reports in the daily papers, have been followed up by correspondence with medical men known to me, and living where the cases were said to have occurred.

The list cannot be considered as a complete one, but my

experience in this sort of work leads me to believe that it may be regarded as fairly representative. From 1888 to 1891, I had access to almost every medical journal published in this country. Since that date—for two and a half years—I have not had the time to push my search for cases so far.

The large number of cases gathered makes it inconvenient to give an account of each, as I did in 1888, and I have therefore tabulated those which have the features which are usually regarded as justifying the diagnosis of hydrophobia, excluding those which, if inserted, would unfairly add to the appearance of absurdity in the commonly accepted notions in regard to hydrophobia.

The period of time covered by these statistics is five and a half years—from June 1, 1888 to December 1, 1893. The total number of cases included in the table is 78. This makes an average of 14 per annum, or about 1 case a year to each $4\frac{1}{3}$ million inhabitants of the United States—about the same proportion which I found at the time of my last report.

The geographical distribution of the cases was as follows: 62 (about 80 per cent.) in the section of country from Maine to Georgia—14 of these being in the New England States; 43 (55 per cent. of the whole number) in New York, New Jersey, Pennsylvania; and 5 altogether in Maryland, South Carolina, and Georgia. Only 16 cases (20 per cent.) occurred in the country west of the Allegheny Mountains.

This distribution I attribute to the influence of the newspapers published in New York and Philadelphia, which have at times, especially those in New York, given too much space to description of the horrors of so-called hydrophobia, and to the exploitation of the Pasteur Institute established in New York. Twenty-four cases occurred in New York City and places in Long Island and New Jersey which may be considered as environs of New York—like Jersey City, Newark, Elizabeth, etc.

The city of Philadelphia furnishes 16 cases of so-called hydrophobia since this subject was last submitted to careful medical discussion. Of these cases, there occurred in 1888,

2 cases; 1889, 1 case; 1890, 4 cases; 1891, 2 cases; 1892, 5 cases; 1893, 2 cases.

The bearing of the fact upon the theory that the prevalence of hydrophobia depends upon the proportion of unscientific to scientific discussion of the subject, seems to me obvious: to others it may not seem so clear.

In regard to the ages at which hydrophobia is said to have occurred, it is interesting to note that of our 78 cases, 18 were under ten years old; 16 were between ten and sixteen years old; 8 were between seventeen and thirty-one years old; 34 were between thirty-five and fifty-seven years old; 1 was said to be "old," and the age of 1 has not been ascertained.

TABLE OF AGES.

Little children.		From 10–16 years.		From 17–31 years.		From 35–57 years.		Old.	Not stated
Baby	1	11 years	5	19 years	2	35 years	2	1?	1?
3 years	2	12 "	4	22 "	3	Adult	23		
4 "	3	13 "	3	27 "	1	40 years	1		
5 "	3	14 "	1	28 "	1	43 "	1		
6 "	3	15 "	2	31 "	1	45 "	1		
6 "	1	16 "	1			48 "	2		
7 years	2					50 "	2		
8 "	2					53 "	1		
9 "	1					57 "	1		
Total	18	Total	16	Total	8	Total	34	1	1

Of these victims of so-called hydrophobia, the only ones too young to have distinct notions in regard to this disease is the "baby" (No. 59), about whom I have no evidence that the case was one of hydrophobia except a newspaper headline; a child of three years (No. 27) who was bitten by a dog that was living and well six months later, when the child died; another child of three years (No. 44) about which I have only a brief newspaper item, saying the disease was pronounced hydrophobia; a child four years old (No. 10) that died in convulsions six weeks after having been bitten by a dog of which nothing was known, and attended by a physician whose name I cannot find in any medical directory; a child four years old (No. 35) that died in Philadelphia after being bitten by a pet dog with

which he was playing and about which there is not a particle of evidence that it was rabid (a case that the coroner refused to admit as hydrophobia); and another child four years old (No. 68), bitten by a dog on whose sore foot he had trodden, and the owner of which scouted the idea that it was rabid.

The other cases of children are within the age when nurses or playmates not seldom talk to them about hydrophobia, and their cases seem to me—for this and other reasons which I cannot now go into—not to furnish any good ground for the common theories as to the nature of this disease.

A study of the relation of the situation of the bite to the age and the length of incubation is interesting. In tabulating them I give no averages, because I regard averages in connection with these points as worthless. The following table contains all the cases in which I have learned these particulars, except two of bites on the scalp and sides.

TABLE OF SITUATION OF BITE, AGE OF PATIENT, AND PERIOD OF INCUBATION.

Face.

Age.	Incubation.	Remarks.
Baby	3 weeks.	
4 years	6 "	Cauterized.
4 "	6 "	"
4 "	8 "	"
4 "	19 "	
5 "	3 "	
6 "	6 "	
6 "	6 "	
8 "	9 "	
9 "	13 "	
11 "	3 "	
13 "	6 "	
Adult	3 "	
"	3 "	
"	6 "	Total 15 cases.

Arm.

14 years	13 weeks.	Cauterized.
Adult	13 "	
40 years	13 "	Total 3 cases.

Leg.

7 years	18 weeks.	
Adult	3 "	
"	13 "	Total 3 cases.

Hand and Wrist.

Age.	Incubation.	Remarks.
5 years	4 weeks.	Cauterized.
11 "	3 "	
11 "	9 "	
12 "	8 "	Dog not rabid.
13 "	19 "	
15 "	several "	
15 "	22 "	
19 "	10 "	
22 "	9 "	
28 "	7 "	Cauterized.
Adult	4 "	
"	5 "	
"	8 "	Cauterized.
"	9 "	
"	13 "	
"	13 "	
"	78 "	
"	12 years.	
35 years	4 weeks.	
48 "	6 "	
48 "	13 "	
50 "	40 "	Cat bite.
53 "	8 "	
Old	13 "	Total 24 cases.

Beside these cases we have one bite on the scalp in a child eleven years old, with an incubation of thirteen weeks, and one on the side in an adult, with an incubation of four weeks.

A study of this table and the two cases just referred to shows the absurdity of the claim repeatedly put forward by Pasteur and his followers, that bites on the face are more dangerous and have a shorter incubation than those on parts more remote from the centre of the nervous system.

We find a child of four years, bitten on the face, with an incubation of nineteen weeks; a man thirty-five years old, bitten on the hand, with an incubation of four weeks; an adult, bitten on the leg, with an incubation of three weeks. The column of bites on the face contains only 5 cases ($\frac{1}{3}$ of the whole) with an incubation of less than the time-honored quota of six weeks; and the column of bites on the hand and wrist contains 6 cases ($\frac{1}{4}$ of the whole) of the same sort. Further analysis of these cases only serves to confirm the belief that there is no warrant for fixing any period as characteristic of the incubation of hydrophobia, or any location of bites as insuring a longer or shorter period of incubation.

After years of careful investigation of this subject, and the accumulation of a very large number of reports, I have no hesitation in saying that the table just given may be regarded as a typical one. Whether we examine fifty cases or thousands, it comes to the same thing. There is no such thing as a characteristic period of incubation. Cases of equal claim to credence are on record with incubations of one day, and of years —for example, Mease speaks of an incubation of less than a day, and Sir William Gull speaks of a case under his observation in which the incubation was thirteen years. Two of our Fellows—Drs. Harrison Allen and H. C. Wood—saw a case which they considered to be hydrophobia after an incubation of three and a half years.

In regard to the period of incubation, as in regard to the relation of the length of incubation to certain details of the bite, averages are delusions. It is absurd to talk about them, because they are equally false to the longest and to the shortest periods, and the probability is that in series of tables an average number would be about the only one which could be hit upon that would never be correct.

My study of hydrophobia for the five and a half years last

past has not served to alter the opinions I have already expressed in regard to the nature of what is called hydrophobia. Investigation dispels many illusions, and what I have done in this subject has given me opinions different from those that I would have entertained had I taken them as those must who have not time to investigate for themselves. I must not ask those who listen to me to agree with my conclusions, but I think after so long, and I trust it will be admitted fairly intelligent and conscientious study, that my opinions are deserving of consideration.

There are several features about this subject that are peculiar to it. One is, that it is so hedged about with superstition and ignorance on the part of medical men. I speak candidly when I say that I cannot find that the average writer on medicine or surgery knows much more about the subject than does the average newspaper writer, and I may say further, that both appear to be about equally disposed to accept apocryphal tales and to report them as true. To show how an error may occur and go on for a good while, I will cite a case which was reported in the *Medical News*, February 8, 1890, which stated that "Principal Clark of the Albion Academy, at Milton, Wis., is reported to have died from hydrophobia, resulting from the bite of a cat ten months previously. The immediate effects of the bite appeared to be trivial, but the hydrophobic symptoms, when they declared themselves recently, were unmistakable and death most painful." On seeing this I wrote to a professional friend living in Beloit (Dr. A. C. Helm), to find out the facts for me. He at once wrote to the President of Milton College, at Milton, and in a few days sent me the reply of President Whitford, which stated that the item was substantially correct. A little later, Dr. Helm sent me a letter written by a sister of Professor Clark, of Albion, in reply to inquiries inaugurated by Dr. Helm and President Whitford, in which she says that her brother "did not die of hydrophobia, as was reported. He died of cerebral meningitis. True," she says, "he had a cat bite, but the cat was not mad, and he had suffered ever since with blood-poisoning, which showed itself in ab-

scesses—he having, in six months, thirty large and small abscesses on hands, arms, and neck. Just how much the cat bite had to do with his death, we cannot tell, but," she adds, "we are very sure he did not die of hydrophobia. There was no attempt to bite, nor aversion to water, but he drank water freely all through his sickness, and enjoyed being bathed as much as any sick person." Thus one case, which may perhaps be classed as authentic in other statistics, melted away in the light of close investigation.

I do not care to burden this report with details which would add needlessly to its length, but I would like to refer to another case which I followed up by correspondence and in other ways. This one was that of a man named Samuel C. Smith, who died in Jersey City, in 1888, and about whom I had most courteous letters from Drs. B. A. Watson, E. W. Pyle, L. A. Opdyke, and Lampson. The story was rather a sensational one. The inoculation was attributed to repeated bites and scratches made by a pet dog during a period of about a year. The victim ridiculed the idea of cauterization, and there is not a scintilla of evidence that the dog was rabid. The patient was manifestly a drinking man and had his first attack of dysphagia while drinking beer. The attack began with convulsive phenomena limited to the thorax and throat. The suggestion of water-drinking either by sound or show excited spasmodic convulsions. The strong morbid condition present was indicated by the fact that the patient required that the keyhole of a cupboard should be filled up "on account of the draught." His disease lasted for five days, at the end of which time he died in coma in spite of the use of a strong faradic current. His treatment appears to have been sufficiently vigorous, and consisted of chloral and (I think) morphine, administered by the rectum and hypodermatically. The man had total suppression of urine for three days before he died.

When we recall these details, and put together the absence of evidence that the man was ever really bitten or that the dog referred to was ever really rabid; that the man was a beer-drinker; that he had manifest morbid conceptions of the effect of draughts of air; the suppression of urine; the energetic

use of narcotics—do you wonder that I regard this as an apocryphal case?

I might briefly allude to a celebrated case occurring at Delanco, New Jersey, in July, 1888.

A man fifty-seven years old, bitten three months before by a strange dog which he was driving away from his own dog, was very nervous; afterward, was caught in a severe storm—an unusually cold day for June—and was chilled and had a fever. While in this state he received a piece of bad news which increased his nervousness, and he began to notice a slight reddening at the point where he had been bitten on the hand. His expressions showed that he had been pursued by the idea of having hydrophobia ever since he was bitten, and that he had thought of going to Paris to be treated by Pasteur. I have certain sources of private information in regard to this case which make it seem to me very plainly one of spurious hydrophobia. I have endeavored to get a scientific statement of the case from the attending physician, but have had no reply to my letter of inquiry.

The whole history of hydrophobia in the United States since June 1, 1888—five years and seven months—includes cases attributed to bites of dogs, cats, men, a spider, a snake; by the inoculation of the blood of a rabid animal, and by the use of the milk of an animal which had been bitten by a rabid animal—in other words it presents specimens of about all the absurdities that ever have been believed in regard to this strange disorder. I have a memorandum of a case reported as hydrophobia in the city of Philadelphia, of a man brought into a hospital, apparently as an alcohol case, who died in a few minutes. The newspaper stated that he died of fear, probably. The Sister in charge wrote me as stated above, and I have never found out anything more about the case. In 1890 there occurred a remarkable case at Columbus, Ohio, where a man who had been brought from a distance of twenty-five miles for treatment, frightened everybody out of the car in which he was, and being refused admission to a hospital, was taken to the jail for the night, where he raved and barked, and afterward died as he was being taken back to the railway station.

This case was published all over the country as one of hydrophobia; but the *Columbus Medical Journal*, of July, 1890, said that all the physicians were agreed that it was not a case of hydrophobia. In 1890 a man died in New York after being bitten, a month before, by a dog about which there was no mention of rabies whatsoever. Having a little difficulty in swallowing, he consulted a doctor, who told him he had hydrophobia, and advised him to go to the Pasteur Institute in New York. By the time he reached New York he was in such a condition that he was taken to Bellevue Hospital. Here the diagnosis was confirmed, his capacity to drink was tested, and he passed into a condition of delirium, being treated with morphine and chloroform, while his struggles were restrained by four men. He had delirium and delusions, and the record states that his temperature was taken frequently, although whenever the thermometer was applied it caused a spasm. He seems to have been visited by a large number of physicians, and so many persons in general, that it is stated the hospital authorities had, finally, to exclude many who came pretending to be doctors. He died suddenly, as those are likely to die who are treated with large and repeated doses of morphine in order to control maniacal manifestations.

In April, 1891, we had a typical occurrence of this sort in Philadelphia. A number of children were bitten by a dog about which little was known, and four weeks afterward one of them, a little girl eleven years old, whose wounds had been "dressed" at the Episcopal Hospital, developed considerable headache and pain in the ears, with convulsions. Physicians were called in, and one is said to have pronounced the case hopeless and to have administered chloroform. Between the convulsive attacks and in the intervals of comparative repose from treatment, the poor child cried out to her mother to kiss her, and said to her, "I won't bite you;" "I'm not mad;" "Please come and kiss me." It is pitiful to read how, under these circumstances, several persons held back the nearly crazed mother on the one hand, and the struggling, longing child on the other. The dog that bit this child was alive when the child died.

About ten years ago I read before the College an account of a case like this, which occurred more than seventy years ago, and I said of that as I say of this, "Picture to yourselves the scene!" Of that case, as of this, my opinion is that it was one of mistaken diagnosis.

In 1884, I published a paper[1] in which I described fully a large number of diseases in which the symptoms of hydrophobia occur: Among them, inflammations of the fauces, causing pain in the ears, and accompanied by nervous manifestations. Just such cases have, in my opinion, occurred many times among children; but when they occur, it happens now and then that there arrives a physician who seems to have no thought of such a thing, who seems to make no thorough examination to exclude all the many diseases which may have symptoms like those of so-called hydrophobia, and which have presented them in cases that I could recount in great number. Such a physician often proceeds, on the assumption of hydrophobia, to treatment which is calculated to raise to the highest pitch the nervous excitement already present, and to deprive his patient of the only chance of life that remains.

And this brings me to a point at which I must for the present stop. I desire to say a word in regard to the treatment of what is called hydrophobia. Some of the Fellows of the College know that I have unremittingly investigated this whole subject for years. I have accumulated a mass of notes which cover thousands of cases; I have studied them carefully; I have had cases under my own care and in consultation; and as a result of all this, I do not hesitate to say that, in my opinion, the treatment recommended in text-books and almost invariably adopted is utterly pernicious. There is no chance that the almost invariable fatality of what is called hydrophobia will be changed until the profession learns that the use of narcotics does harm and never does good.

To describe the mode of treatment of hydrophobia more

[1] "Disorders Mistaken for Hydrophobia." Trans. Med. Soc. of the State of Pennsylvania, 1884.

likely to do good, I shall borrow and adopt some suggestions that I made before the American Medical Association in 1884.[1]

The first thing to be done is to bring some sort of order out of the confusion which usually exists before the doctor arrives. Well-meaning, but hurtful, jailers must be called off from the patient, the horrified and horrifying spectators must be dispersed, the patient must be placed in as quiet surroundings as possible, while the doctor strengthens those about by himself setting an example of steady composure. A tolerable degree of quiet having been secured, the next step must be to ascertain what is the real nature of the disorder with which the patient is afflicted. I have elsewhere shown[2] that there is a large variety of organic and functional diseases having no connection with the bite of a rabid animal, in which the symptoms of hydrophobia are presented. The physician should be familiar with all these and search for their presence with a most rigid examination; and only when all possibility of their presence has been excluded should the diagnosis be concluded upon as one of hydrophobia. If any one of these simulating disorders be found to be the cause of the symptoms, of course the treatment should be that which is proper to that particular disorder. Into the details of this we may not now go.

But, if the most painstaking and intelligent investigation leaves no alternative but to conclude that the disorder is indeed what is generally understood by the term hydrophobia, then a plan of treatment must be adopted which corresponds with what is known in regard to the nature of this disorder. Such a plan includes certain general—we may say, moral—principles as well as a *modus medendi*. Indeed, I believe the former are of a greater importance, in our present state of knowledge, than the latter. The first general principle to be observed in the management of a case of supposed hydrophobia is not to be misled by the real or apparent inability of the patient to drink; and joined with this should be an attempt to prevent the patient from being misled by it. The doctor should know

[1] "The Treatment of Hydrophobia Historically and Practically Considered." Journ. of the Amer. Med. Assoc., August 16, 1884.

[2] "Disorders Mistaken for Hydrophobia."

that this symptom, in the very manner and form supposed to be peculiar to hydrophobia, may be absent in this and present in a number of other disorders. The patient should be also informed of this fact in every case. Further, both doctor and patient should get rid of the time-honored but utterly erroneous notion that the patient's life is endangered by this inability: and, since this is so, all attempts to induce a patient to drink are useless at the best and pernicious at the worst. As a means of aiding the diagnosis they are of no use to the physician, and they always do harm to the sufferer. If this sufferer be a little child, and force be used, as it too often has been, to compel the patient to drink, then the perniciousness of the practice rises to the point of absolute cruelty, and marks at once the ignorance and the hardness of the doctor.

Only less reprehensible than this are such tests as presenting a looking-glass to the patient, or blowing or sprinkling water on him, which ought to be forever banished from an enlightened medical practice.

Almost every writer on the subject of hydrophobia warns against the danger of making inquiry, in the hearing of a patient, after a suspected bite. I think that one might even go farther than this, and say that inquiry after a bite of a mad creature may well be postponed altogether until after the death or recovery of the patient. The knowledge of such a bite is never necessary to the diagnosis, and the determination that no such a bite has occurred does not overthrow it. Such inquiries always do harm to the patient or his friends, and they never do any good to the doctor, at that time. He may therefore well exercise his patience and restrain his curiosity until his patient is recovered or dead, when an exhaustive examination of all the elements of the case may contribute to the advance of science without prejudice to the subject of the investigation.

But, while observing the precautions just mentioned, I think one may err in preserving too scrupulous a silence or insincerely repudiating the idea of hydrophobia. If this idea really has not entered the head of the patient, then it ought by no means to be suggested. But if it be already there, the subject must not be so studiously avoided—much less made the occa-

sion of lying—as to betray to the patient the despair with which it fills the doctor. The latter need never deceive himself with the notion that he can thus hide the dreadful conviction which has taken possession of him. When matters have come to this pass, frankness and as much fearlessness as can be summoned are best calculated to win the sufferer's confidence.

On the other hand, while the doctor is avoiding the useless attempt to deceive his patient, by a too significant silence, as to the apparent character of the disorder, he should not be misled by a corresponding attempt on the part of the patient. It is far from uncommon for the latter, by persistent silence or even by stout, and seemingly sincere denials, to strive to convince himself as well as his physician that he cannot have hydrophobia. For such is the strangeness of this disorder that, as the Spartan youth denied all knowledge of the stolen fox hid under his cloak and at that moment tearing at his vitals, so in cases of supposed hydrophobia will both patient and doctor try to deceive each other as to what is going on in their minds, the one to save his life, the other to redeem it, until each sees that the other has recognized the poor attempt, and both give it up before the horror which will no longer be held down.

Again, the sufferer with hydrophobia should never be treated with violence. If the disorder be an acute mania, or the fury of a convulsive or febrile delirium, forcible restraint may be required. But it is not needed in hydrophobia. It is true that patients sometimes call for it, and doctors often prescribe it. But in both instances this shows the possession of popular, as opposed to scientific, ideas in regard to the subject. Forcible restraint is not demanded for the safety of the bystanders, and it is injurious to the patient Indeed, there can be no doubt that the greatest struggles of patients who have been subjected to force has been provoked by that very force. The fear of ordinary acts of violence on the part of one supposed to have hydrophobia is usually supplemented by the fear that a like disorder may be contracted from his bite or the absorption in any way of his saliva. This fear is utterly groundless. There is not one respectable assertion in all the literature of the subject in support of it. I have myself had occasion to put this

confidence to the proof, and I have no uncertainty in regard to it. I recommend the attainment of a like confidence to others. For they must be without fear themselves if they would encourage others. And if they have no fear, then they can be of great service at a time when fearlessness seems to be about all that is left to those who would be helpful if they could.

Thus every effort of the doctor must be used to obtain and preserve as great a degree of calm as possible. Then the patient must be disturbed as little as possible. No offer of food or drink should be made. For if a man can live forty days without eating, one may well take the responsibility of yielding to the determination of a patient who not only fears and hates the thought of meats and drinks, but spews them out if one succeeds in forcing them upon him.

As to the medicinal treatment of hydrophobia—the less there is of this the better. No medicine at all is better than any medicine that cannot be administered without seriously disturbing the patient. In most cases there are three ways of administering drugs which are available: by the rectum, by inunction, and by hypodermatic injection. In many cases medicines can at times be administered by the mouth. In choosing his remedies and deciding upon the quantity to be given, the practitioner must not be tempted into meeting the violence of the disorder with opposing violence of medication. Whatever plan of treatment be adopted it must be temperate. No use of large doses of morphine, chloral, atropine, curare, chloroform, or such-like, has ever proved of value, and I am convinced that these drugs have killed many patients. Here, if ever, it is the "soft answer" which may be expected to "turn away wrath." I think the use of moderate doses of cannabis Indica may prove useful. Alcohol, in small and repeated doses, taken in hot water, I think might prove a valuable remedy—it being understood that I presume the exclusion of an inflammatory disorder with hydrophobia merely as a concomitant. I am inclined to think also that something might be expected of the method of "mixed narcosis," proposed for surgical operations, in 1877, by Nussbaum. In this method a dose of $\frac{1}{4}$ to $\frac{1}{2}$ grain of morphine is injected hypodermically, and in a few minutes

chloroform is given by inhalation. If this could be done without too great resistance on the part of a patient with hydrophobia, I believe it would prove very advantageous. The use of prolonged hot baths I believe may do good—not as a specific, as some have foolishly supposed, but by the activity of the circulation they produce and their invigorating effect. Finally, I would suggest the use of the hydrobromate of hyoscine, which has proved of great value in maniacal neuroses. I have had no opportunity to use it in hydrophobia, since its employment has been fully tested; but I certainly should try it if I had.

In all the treatment of hydrophobia, the cardinal principle should be that the medication must be reduced to a minimum. Indeed, I have no doubt that better results than have been attained by any method heretofore recommended would be secured if no medication at all were employed. There can be no doubt that it is true, as Decroix asserts, that the ordinary methods of treatment act adversely to the interests of the patient, and that the rational plan is restricted to two indications: to protect the patients from all causes of excitation; to procure for them all the moral and material satisfactions compatible with their state.[1] "By adopting this method," Decroix adds, "there will be obtained a natural hydrophobia more benign than the artificial hydrophobia, which is a consequence of incendiary medication, and cases of cure will certainly be less rare than in the past."

In 1883 M. Barthélemy called attention to the suggestions of Decroix, and emphasized them by a reference to the practice of M. Abadie, a veterinarian of the Department of the Loire Inférieure, in the treatment of tetanus in horses. The latter, after observing the lamentable failure of active treatment, adopted with marked success the plan of leaving the animals strictly alone in a darkened room, from which all sights and sounds were rigidly excluded. Further, M. Barthélemy recalls the method adopted by M. le Docteur Bouley[2] to

[1] Decroix, M. E.: "Note sur la Curabilité de la Rage." Bull. de l'Acad. de Méd., 20 Juin, 1882, p. 699.

[2] This physician must not be confounded with M. Henri Bouley, the veterinarian, who is famous for his studies of rabies.

control violently hysterical patients. This consisted in shutting them up in a padded room from which all light was excluded. "C'était," he says, "ce que notre maître appelait le traitement par astimulation." From this he thinks a profitable lesson may be drawn as to the management of cases of hydrophobia.[1] This is undoubtedly true, and it seems strange that the obvious deduction from the success which has often followed the administration of absurd and inefficient nostrums—namely, that no medication is better than active treatment—has failed to secure the application in teaching and in practice which it should have had.

I have elsewhere stated the conviction to which observation and a laborious study of this subject have led me, namely, that hydrophobia is not a specific, inoculable disease; and that the dread of swallowing, and the other phenomena which are known as hydrophobia, do not indicate a specific morbid process, but are mere symptoms of a variety of disturbances of the animal economy, some of these being of an organic, others of a purely functional (in the usual sense of this term) character.[2] I do not deny that there is a set of symptoms which for want of a better name may, without great impropriety, be called "hydrophobia." What I deny is that these symptoms depend upon a specific virus derived from a rabid animal. It is undeniable that the symptoms of hydrophobia may *follow* the bite of a dog suffering with what is called rabies; but so they may, in most typical form, the bite of one that is simply angry, or not angry at all, and quite healthy, or of a man who is simply angry, or they may arise in the progress of a variety of diseases, or spontaneously as the result of fear. In all these cases the phenomena and the issue are exactly the same. There can, therefore, be nothing specific in the saliva of a so-called mad dog, unless the word "specific" be given a new meaning to suit this particular case.

[1] Barthélemy: Le Hoang-nan et le Rage. Bull. gén. de Thérapeutique, t. cv., 1883, pp. 147–160.

[2] See Trans. of the College of Physicians of Philadelphia, 3d ser., vol. vii. pp. 17–35. New York Med. Journal, Dec. 29, 1883, vol. xxxviii. pp. 707–711.

TABLE OF CASES IN THE UNITED STATES FROM JUNE 1, 1888, TO JANUARY 1, 1894.

No.	Date of death.	Place.	Sex and age.	Animal.	Description.	Location of bite.	Incubation.	Mania, dog symptoms, etc.	Restraint.	Narcotics used.	Remarks.
	1888										
1	June 6	Jersey City, N. J.	M. 50	Dog	Pet, not rabid.	.....	4 weeks	.....	.....	Chloral.	Drinking man; often scratched by dog's teeth for 1½ years. Suppression of urine. Stuffed up keyhole of *cupboard*. Very nervous after the bite.
2	July 2	Delanco, N. J.	M. 57	"	Attacked; killed.	.....	3 mos.	Tried to eat grass.	.....	.....	Feared hydrophobia.
3	July 6	Fairfield, S. C	M. 22	Cat	Strange; killed.	.....	9 mos.	Begged to be killed.	Tied hand and foot.		
4	July 22	Chicago	F. 28	Dog	Wounded by strange dog.	Hand	7 weeks	Maniacal.	.....	Chloral, stramonium, whisky by mouth and rectum.	Bite cauterized. Rheumatism of left shoulder. Prompt diagnosis. Physicians lied to her and she lied to them.
5	Aug. 18	Camden, N. J.	M. 13	"	Strange.	Face	6 weeks	.....	Three men held.	Morphine.	Bite cauterized.
6	Aug. 30	Madison, N. J.	M. 7	"	Persecuted	.....	2 mos.	.....	.....	.....	Newark physician pronounced hopeless.
7	Oct. 23	Jersey City, N. J.	M. 31	"	. .....	.....	1 mo.	Maniacal, barked and bit.	Policemen	.....	Prompt diagnosis.
8	Dec. 14	Philadelphia	M. 8	"	Unknown.	Cheek and lip.	2 mos.	Desire to injure.	.....	Salicylate of soda and morphine.	Bite cauterized. As soon as bitten, men told boy he had hydrophobia.
9	Dec. 28	St. Louis	M. ad't	"	.....	Leg	3 mos.				
10	Dec. 31	Philadelphia	M. 4	"	Strange.	Cheek	6 weeks				
	1889										
11	Jan. 1	Fall River, Mass.	M. ad't	"	.....	Finger	3 mos	.....	.....	Ether, morphine, curare.	Denied the bite when asked. Expected to die. "Stone met his death like a hero."

12	Jan. 18	Elizabeth, N. J.	M. ad't	Dog	.....	Thumb	3 mos.? 10 days?				
13	Feb. 6	Palatine, Ill.	M. 45	"	Rabid.	Face	6 weeks	. .....	.....	.....	Bite cauterized. Madstone used.
14	Feb. 24	South Portsmouth, R.I.	F. lt'le	"	.....	.....	5 mos.				
15	Feb. 28	Wooster, O.	M. 11	"	.....	.....	8 years	Bit off two of his own fingers.	.....	.....	Said to be "dying of hydrophobia."
16	Feb. 28	Flatbush	F. 9½	"	Killed.	Face	3 mos.	.....	....	.....	Bite cauterized. Pain in chest, choking, convulsions, death.
17	Apr. 12	Scituate, Mass	M. 22	"	Savage.	.....	6 mos.				
18	June 21	Long Island City	M. 11	"	Rabid.	Finger	9 weeks	Snarled and bit.	.....	.....	Symptoms lasted four weeks; doctors said *not* hydrophobia.
19	Aug. 13	Philadelphia	F. 48	"	Alive when patient was dead.	Hand	6 weeks	.....	....	. ...	Pain attributed to rheumatism.
20	Oct. 20 1890	Astoria, L. I.	M. ad't	"	.....	.....	6 mos.	Frothed and bit	.....	.....	Saloon-keeper.
21	Jan. 25	Sabatba, Kan.	M. ad't	Man	.....	Finger	2 mos.	.....	....	....	"Exhibited every symptom of hydrophobia."
22	Feb. 17	Newark, N. J.	M. 35	Dog	.....	Hand	2 years	Frothed and bit.	Four men held.	.....	
23	Apr. 9	Boston	M. 35	"	Sick.	Finger	4 weeks	Maniacal.	Strait-jacket.	Bromide of potash, morphine, chloral.	Fixed idea that physicians intended to do him bodily harm. Had diarrhœa for a week; delirious; vomited clotted blood.
24	Apr. 13	Watertown, Mass.	M. 7	"	Not rabid	Leg	4½ mos.	Frothed.	.....		
25	May 10	Woburn, Mass.	F. 13	"	Rabid.	.....	1 year				
26	May	Lewistown, Ill.	M. ad't	"	....	.....	.....	.....	Chained to floor.	.....	Madstone used; had himself chained to floor.
27	June 8	Peoria, Ill.	M. 2	"	Not rabid.	.....	6 mos.	Maniacal; bit his mother and aunt.			

No.	Date of death.	Place.	Sex and age.	Animal.	Description.	Location of bite.	Incubation.	Mania, dog symptoms, etc.	Restraint.	Narcotics used.	Remarks.
28	June 10	New York	M. ad't	Dog	Not rabid; killed.	Wrist	1 mo.	Wildly maniacal.	Four men bound him	Chlorof'm and morphine.	Had pain in scar; doctor told him he had hydrophobia; sent him to N. Y. Pasteur Institute, too late; to Bellevue Hospital; many doctors and others came to see his symptoms.
29	June 25	Brunswick, Me.	M. ad't	"							
30	June 26	Woburn, Mass.	M 11	"	.....	.....	2 mos.				
31	July 2	Newark, N. J.	M. 12	"	.....	.....	.....	Frothed and snapped.	.....	.....	Broke two ribs two days before outbreak.
32	July 2	Philadelphia	M. 6	"	Strange.	Cheek	6 weeks	.....	.....	Chlorof'm and morphine	Cough like bark of a dog.
33	July 16	Lowell, Mass.	M. 16	"	.....	.....	3 mos.				
34	July	Pawtucket, R. I.	M. ad't	"	.....	Wrist	2 mos.	Maniacal.	.....	.....	Said to be "dying."
35	Aug. 1	Philadelphia	M. 4	"	Strange.	Cheek	4 mos.	.....	.....	.....	Physician used "remedies used by Pasteur." Coroner said *not* hydrophobia.
36	Aug. 1	Newark, N. J.	M. 12	"	Not rabid.	Hand	2 mos.				
37	Sept. 5	Boston	M. 53	"	Fighting.	Thumb	2 mos.	Maniacal, tried to bite attendants	.....	Chloral, chlorof'm, bromide of potash.	Violent vomiting; convulsion; left arm paralyzed. Rabbits inoculated successfully.
38	Sept. 8	Philadelphia	M. 5	"	Persecuted	Hand	1 mo.	.....	.....	"All remedies known."	

39	Oct. 4	Columbus, O.	M. 12	Dog	Vicious; killed.	.....	4 mos.				
40	Oct. 16	Atlanta, Ga.	M. ad't	"	.....	.....	6 mos.	.....	Seven men held him.	Opiate.	Eight men piled a mattress on him; doctor injected an opiate, and he "passed away."
41	Nov. 21	Philadelphia	M. 13	"	Killed.	Hand	4 mos.	.....	.....	Powerful opiates.	
42	Nov. 22 1891	Hackensack, N. J.	M. 22	"	Rabid.	Hand	9 weeks	.....	.....	.....	Cigarette smoker.
43	Jan. 12	Chicago	F. ad't	"	.....	Face	6 weeks	Maniacal; bit and fought.	Police held her.	Morphine, cocaine, chlorof'm.	Pregnant. Told to go to Chicago Pasteur Institute. Soon to be confined.
44	Jan. 12	Indianapolis Ind.	F. 3	"	.....	Scalp	3 mos.	.....	.....	.....	Moaned, screamed, convulsed; eyes open for 24 hours, and corneæ dried.
45	Jan. 23	New York	M. 27	"	.....	.....	.....	.....	Securely pinioned.		
46	Feb. 20	Rockville, Conn.	F. 48	Cat	.....	Finger	3 mos.	.....	.....	.....	"Physicians think she will die."
47	?	Paoli, Pa.	M. ad't	...	... .	.....	6 mos.	Frothed.	Five men held him.		
48	Apr. 27	Philadelphia	F. 11	Dog	Living yet	Cheek	3 weeks	Maniacal.	Three persons held her.	Chlorof'm	Headache, pain in ears, spasms; cried out, "I'm not mad."
49	?	Philadelphia	M. 14	"	Strange.	Arm	3 mos.	Barking and biting.	Four men held him.	.....	Doctors say "no hope of recovery."
50	June 6	Asbury Park N. J.	M. 50	Cat	Strange.	Finger	9 mos.	.....	Two men held him.	"Powerful opiates."	Bartine. Read up on hydrophobia; said he had it and would die. His fiancée assisted at autopsy. Made his will, and waited to die.
51	June 25	Rutledge, Ga.	M. ad't	Dog	Attacked man.	Face	3 weeks	.....	.....	.....	Feared hydrophobia.
52	?	Saginaw, Mich.	M.	"	Strange.	.....	4 weeks	Maniacal; barks and snarls.	Three or four men held him.	.....	Death expected.
53	July 30	Wichita, Kan.	M. 12	...	.....	.....	Months	Maniacal; barks and snarls.	Four men held him.	.....	See No. 73.

No.	Date of death.	Place.	Sex and age.	Animal.	Description.	Location of bite.	Incubation.	Mania, dog symptoms, etc.	Restraint.	Narcotics used.	Remarks.
54	Aug. 9	Ashland, Mass.	M. 5	Dog	Strange.	Face and hands.	3 weeks	.....	.....	.....	Bite cauterized. To N. Y. Pasteur Institute next day. *Cured* in sixteen days. Six days later stupor and nausea; one pupil dilated; fever, twitching, delirium, convulsions, phlegm in throat, concentric eruption, death. Two sisters bitten at same time lived.
55	Oct. 11	Elizabeth, N. J.	M. 8	"	.....	.....	10 weeks	.....	.....	Narcotics.	
56	Oct. 25	Columbia, S. C.	M. ad't	"	Rabid.	Arm	3 mos.	Acting like a dog.	.....	....	Pain in arm where bitten and side where inoculated. Treated and "cured" at N. Y. Pasteur Institute.
57	Nov. 9 1892	Trenton, N. J.	M. 5	"	... .	.....	3 mos.	.....	.....	.....	"Two brothers bitten at same time are ill."
58	Jan. 1	Monroe, N. J.	M. 40	"	Strange.	Arm	3 mos.	.....	Six men held him.	.....	Feared hydrophobia. Had been treated at N. Y. Pasteur Institute.
59	Jan. 3	Newark, N. J.	b'by	"	.....	Head and face.	3 weeks				
60	Feb. 2	Baltimore	M. ad't	"	.....	.....	6 weeks				
61	?	Cooperstown Pa.	M. ad't	"	.....	Leg	3 weeks	Tied to settee and bit it.	Tied down	.....	Paralyzed left leg and arm. Said he might bite, and was tied down.
62	Feb. 20	Philadelphia	M. 15	"	Pet, in fits	Wrist	7 mos.	.....	Held by several persons.	"Hypodermics."	Father died of hydrophobia a year later. See No. 76.
63	Mar. 29	Arkansas	M. ad't	"	Rabid.	Wrist	12 years				
64	Apr. 14	Trenton	M. ad't	"	.....	.....	Few days	.....	.....	.....	A dog-fancier.
65	May 30	Philadelphia	M. 4	"	Sore foot; killed	Cheek	weeks.	.....	Several men held.	.....	See case No. 67.

66	June 8	Lynn, Mass.	M. ad't	Dog	.....	Lip	3 weeks	Bit and chewed wood of his bed.	.....	.....	Death attributed to fright.
67	June 27	Philadelphia	F. old	"	Sore foot; killed.	Hand	3 mos.	Asked son to shoot her.	Held.	.....	See case No. 65. Diagnosis immediate.
68	?	Philadelphia	M. 11	"	.....	Finger	3 weeks	.....	.....	.....	"Will probably die."
69	Aug. 1	Philadelphia	M. 19	"	.....	.....	3 mos.	.....	.....	Every remedy.	Abscess under arm. Jaws stiff.
70	Aug. 14	Chicago	M. 6	"	.....	.....	.....	.....	.....	.....	Had been refused treatment by Lozario.
71	Sept.	Ashbourne, Pa.	M. 15	"	Fits.	Wrist	Several weeks	.....	.....	Quieting hypodermatics.	Physician made diagnosis of hydrophobia at once. Arm paralyzed, mouth set.
72	Oct. 5	Morrisania, N. J.	M. ad't	"	....	Finger	5 weeks				
73	Oct. 6	Wichita, Kan.	M. ad't	"	Rabid.	Side	4 weeks	.....	.....	.....	See No. 53.
74	Dec. 5	Jersey City, N. J.	M. 6	"	Tail amp. and cauterized.	Head and face.	6 weeks	.....	.....	Chloral and bromides.	
75	Feb. 4	Leav'nworth Kan.	M. ad't	"	Rabid.	.....	6 mos.	Tried to bite.	Restrained	.....	Madstone used.
76	Feb. 24	Philadelphia	M.	"	Fits; no thought of rabies.	Thumb	18 mos.	.....	Three men held him.	.....	Three others bitten in family. No. 62 died Feb. 20, 1892. Man said he would die Friday, and he did.
77	July 17	Asbury Park, N. J.	F. 19	"	Pet.	Hand	2½ mos.	Bit and frothed, and scratched her lover.	Four men held her.	Morphine, powerful injections.	Not a bite, but a mark of tooth. Had a heavy cold. Companions had asked her about hydrophobia.
78	Oct. 28	Philadelphia	M. 43	"	Still alive.	.....	8 mos.	Maniacal; drove his wife out of doors.	.....	Morphine, large amount.	Bite cauterized. Questions and newspaper clippings about hydrophobia drove him mad.

DISCUSSION.

Dr. A. C. Abbott: I think the College should feel itself indebted to Dr. Dulles for this summary of a group of most interesting cases, which to me, however, have more the appearance of hysteria than of hydrophobia. The histories, as given by Dr. Dulles, are in the main derived from newspapers, and are the sort of descriptions generally given of these cases by those who view only their clinical aspect. Clinicians who have seen both the true and pseudo-hydrophobia agree that it is often difficult to distinguish between them without a complete antecedent history.

Of all the cases mentioned by Dr. Dulles this evening, not one of them presented a history of bite by a rabid animal. And from what he has said, one might be led to conclude that there is no such disease in man as hydrophobia following upon the bite of animals affected with rabies; in fact, he states that in his opinion there is no such specific disease as hydrophobia in man. If this be true it is difficult to comprehend what the nature of the disease is upon which Pasteur has done such brilliant work. If we accept his published results, and there is no reason for doing otherwise, he *must be* working with a specific disease; a disease that is transmissible from animal to animal through the bite, and which is accompanied by a definite train of symptoms, can hardly be conceived as being other than specific and infectious.

Among the results of Pasteur's work it has been shown that rabies is not confined to dogs, but is often encountered in cats, wolves, hogs, and cattle; that it is transmissible from one species to another through biting, and that it manifests itself clinically in two forms—the violent and paralytic rabies. Examination of the central nervous system of fatal cases reveals the fact that it is not a disease without demonstrable pathological lesions; lesions, it is true, that are hardly to be considered as characteristic, but, nevertheless, lesions that accompany practically all cases.

What is of still more importance is his demonstration of a specific *something* in the central nervous system of rabid animals. If a dog suffering from the symptoms of rabies is killed and a portion of the medulla of that dog is introduced into the cranial cavity of a rabbit, that rabbit dies with a certain definite group of symptoms. If from this rabbit a second rabbit is inoculated, it also dies with the same group of symptoms. These symptoms have a period of incubation of twelve to fifteen days. If the experiment is continued through ten or twelve rabbits the period of incubation diminishes, until ultimately it reaches a point that may be called the minimum, namely, six days. When this period is reached successive inoculation in a hundred rabbits does not change it. If at any time during the experiments, the medulla of the rabbit is introduced into the dog, the typical group of symptoms is reproduced.

We can say nothing as to the nature of the virus. We cannot say that it is a bacterium, and we think that it is not, though in many respects its behavior is closely analogous to that of bacteria. The virulence of the poison is destroyed in a few minutes by exposing the medulla to a temperature of 50° C. It is destroyed by exposure to corrosive sublimate and to other chemical disinfectants. It is also destroyed by drying.

It has been said that hydrophobia has no pathology. Possibly it has not a characteristic pathology, but pathological lesions unquestionably accompany the hydrophobic process. It has been repeatedly found in human beings bitten by dogs known to have had rabies, that there is congestion of the medulla about the roots of the cranial nerves, with small hemorrhages and accumulations of lymph cells. There is also accumulation of similar cells in the nerve substance, and thrombi of a hyaline character in the small vessels. While these are not absolutely characteristic, the fact that they have been repeatedly found in these cases looks very much as though they were the result of the causative agent.

The most important point is with reference to the results of preventive inoculation. The general statistics, as I recall them, of the fatality among individuals who have been bitten by dogs known to have had rabies is 15 per cent., while the mortality among those bitten on *exposed* surfaces is about 60 per cent. This mortality has been reduced, according to Pasteur's statistics, which we unhesitatingly accept, to between 1 and 3 per cent.

Dr. Dulles referred to the abundance of literature on the subject in France. One of the reasons why we have so much literature on this subject from France is because they have no dog laws. Dogs are kept by almost everyone, and are allowed to run at large unmuzzled and unattended. Next door to France, in Germany, there are rigid dog laws: in Germany hydrophobia is one of the rarest diseases, while in Paris it is, as we know, comparatively frequent.

Dr. John Ashhurst, Jr.: My personal experience with hydrophobia is limited to one case which I saw in consultation some years ago. The patient promptly died, as these patients ordinarily do. I should not have arisen to say anything this evening, but it seemed to me that the remarks of Dr. Abbott might give rise to an erroneous impression in regard to a practical point, and that is with reference to the so-called prophylactic treatment. I think that there is no question at all that there is such a disease as hydrophobia, and such a disease as rabies in the dog, with its two varieties—the paralytic and the furious form. It is not proven, however, that hydrophobia in man is precisely the same as rabies in the dog. I have no doubt at all as to the correctness of the experiments and observations of M. Pasteur as to the propagation of the disease from one animal to another, nor as to the fact that by propagating the disease through certain animals its virulence can be intensified, while by propagating it through other animals its virulence can be diminished, so that the spinal cords first

used in the prophylactic treatment are practically inert; but when we come to the application of these facts to the treatment of a person bitten by a dog supposed to be rabid, a false inference may be drawn. As a matter of fact, hydrophobia is a very rare affection. In a considerable hospital experience, extending over thirty years, I have seen but one patient with hydrophobia. I do not know of any medical man in this neighborhood who has seen more than one or two cases. Dr. Dulles may have seen more, as he is in a position to be called to these cases. The records of London for a number of years prior to Pasteur's researches showed the mortality to be about two and a half patients annually, or five in two years. Now, if we look at the mortality in places where establishments for the prophylactic treatment have been founded, we find that while there is a smaller percentage of deaths, yet the absolute mortality in number of cases is increased very considerably. It seems to me that the physician should be very sure that the patient is threatened with hydrophobia, as the result of inoculation of rabies from a lower animal, before he recommends the prophylactic treatment.

I have not the same confidence in the clinical results of Pasteur's studies that I have in his pathological observations. There seems to be no doubt that patients have been discharged from Pasteur institutes as cured, who have afterward developed hydrophobia, and in some of these cases there have been grounds for at least suspicion, if not more than suspicion, that the disease has been caused by the prophylactic inoculations themselves and not by the original bite. Under these circumstances the surgeon should be very certain indeed that the patient is in danger before recommending the treatment. We all know that dog-bites are very common. In a large hospital scarcely a week passes without two or three such cases coming under observation. The usual plan of treatment is that recommended by Mr. Youatt—cauterization with nitrate of silver. Whether or not this has any other effect than reassuring the patient I do not know, but hydrophobia almost never follows. Although negative evidence of this kind is, of course, of little value as compared with positive evidence, yet it has a certain weight.

The fact that so many dog-bites are harmless, even when the dog is supposed to be rabid, and the fact that hydrophobia is such a rare disease, should make the surgeon very certain indeed that the patient is in danger before he recommends this so-called prophylactic treatment, which is by no means always successful, and which sometimes seems to cause the very disease it is meant to prevent.

THE PRESIDENT: What would Dr. Ashhurst do under circumstances of this kind: A person is bitten on a bare surface, and the head of the dog is taken and inoculations made into rabbits, with the development of the disease in twelve days? Such a case has recently occurred in Baltimore. A young man was bitten by a dog; he killed the dog and took it home and

sent the head to Dr. Welch. The spinal cord was inoculated into rabbits and they developed hydrophobia. The young man has gone to the Pasteur Institute in New York

Dr. Ashhurst: In a case such as the President cites I should endeavor to set all the facts before the patient and let him decide whether or not he would submit to this treatment. I do not mean to say that prophylactic treatment is never justifiable, but that there is strong reason to believe that it has, upon the whole, done more harm than good.

Dr. Abbott: I must take exception to the remark of Dr. Ashhurst, viz., "that prophylactic treatment has done more harm than good." Just what the proportion of deaths after treatment is in Paris I cannot at the moment say, but it is low. I believe that there have been some deaths in other institutions. I know that the deaths which have occurred after inoculation have been very few and not sufficient to bring the mortality up to what it would have been if these cases had not been treated.

Dr. Charles W. Dulles: This is a very large subject for discussion, and I intended to give this evening only certain deductions from the cases that I have been studying in the last five years. I think that Dr. Abbott slightly misunderstood my paper. My table contains seventy-eight cases, but I gave an epitome of only a few to bring out certain points in regard to the subject. His supposition that I have collected cases that no one would regard as hydrophobia is erroneous. As I said in my paper, I excluded from my table cases that would have unfairly added to the appearance of absurdity, and put in it only those that come up to the common picture of the disease.

My opinions in regard to hydrophobia are based upon unremitting and long-continued study; and I may state that there has not been an objection to them offered to-night that I have not heard over and over again, and that I have not carefully considered in my own mind. Dr. Ashhurst's remarks imply that there is a similarity between rabies in dogs and hydrophobia in man. I have in the past carefully avoided discussing publicly the subject of rabies in the lower animals—not because I have not studied it, but because to have brought it in would have only made confusion worse confounded. But I may call attention to the fact that there is no similarity between rabies in the dog and hydrophobia in man. There is hardly a point in which they are alike. The man dreads water—the dog flies to water; the man is hyperæsthetic—the dog is anæsthetic—you can beat him with a red-hot bar of iron and he does not seem to feel it.

With regard to the occurrence of rabies in dogs, I have some notes. Among them is the fact that, at the London Home for Lost Dogs, among 200,000 dogs brought to the institution, there was not one case of rabies. I know a number of persons who have the care of dogs and have charge of kennels, who have never seen a dog with rabies. But I cannot to-night go over the evidence bearing on this point.

In view of the absolute and remarkable faith which some medical men have in Pasteur's statements about hydrophobia, I would say that I know Pasteur's history from the time when he began his career. I have the greatest admiration for the good things that he has done and for his wonderful ingenuity and resources; but I think that he knows very little on the subject of hydrophobia. In regard to it he has made most positive statements which he has had to take back. I went over this subject in detail before the Philadelphia County Medical Society, in a paper published in the *Medical Record*, February 13, 1886; and my paper is open to anybody who wishes to take it up and discuss it.

Dr. Abbott has uncommon familiarity with the bacterial side of this subject, but when he says that the artificial virus of Pasteur is such a singular virus that it demonstrates its peculiarity by producing in animals at a "certain" period "certain" symptoms, then he shows that he is not familiar with the entire history of experimental rabies. Dr. Ernst, of Boston, made the same sort of statement in this room before the Academy of Surgery, some years ago; and I then showed that his own experiments contradicted it. There is no such thing as a uniform and typical series of manifestations after inoculation with the medullæ of artificial rabies.

Furthermore, this whole idea that the medulla of a rabid animal contains—more than any other tissue—the virus of rabies, is contradicted by hosts of published experiments of which Pasteur and his followers seem to be ignorant, and is a gratuitous appropriation of an ingenious but erroneous theory enunciated by Davaine before Pasteur took up the study.

With regard to statistics, I cannot go into details now; but I may say that Pasteur's statistics would indicate that the liability to death from hydrophobia in France has multiplied enormously since he began his inoculations. The figures are about these: during the last five years when we had in the United States about 14 cases a year, Pasteur is said, by his enthusiastic admirers, to have saved from death 8581 persons. And during this time the actual number of deaths from hydrophobia have increased in France. If to the actual deaths we add the number of presumptive deaths used to prepare the so-called death-rate of the statistics of the Pasteur Institute, the whole number would be absolutely incredible.

Dr. Abbott has said that the reason that they have so much hydrophobia in France, is because dogs run at large there, and because France has no effective dog laws. Do not dogs run at large in the city of Constantinople, and a great deal worse dogs? Yet there is so little hydrophobia in Constantinople that some writers have said that rabies never occurs there. On the other hand, it has been said that hydrophobia is practically unknown in Germany, because the dogs there are muzzled. This is another mistake; dogs in Germany are not uniformly and effectively muzzled everywhere, and hydrophobia occurs there, although not nearly so often as in France.

It is said that Pasteur has reduced the mortality from hydrophobia to

one-half of one per cent. One way in which this preposterous claim is worked up is to be seen in the fact that Vulpian had the audacity to state before the Académie de Médecine, that the ordinary mortality after bites by rabid animals is sixteen per cent. If he knew anything of this subject he knew that there is no such thing as an average mortality. No statistics fix such a figure. The most frequently quoted and adopted statement in regard to this goes back to Hunter's time, who speaks of twenty persons bitten, with one case of hydrophobia, a mortality of five per cent.—not sixteen per cent. It is surprising to see the contradictions in which those are involved who have run after the theories of Pasteur. In 1884 Gibier (now of New York) published a book—which is in the College library—in which he describes and pictures the micrococcus of rabies, and gives an account of experiments in which he proves its specific character. But before long he dropped this interesting work and came to this country to found the Pasteur Institute in New York.

Pasteur himself found and described a germ of rabies, but soon had to take it back. When Colin, of Alfort, in the Académie de Médecine, said "this organism was not new or peculiar to rabies and that Pasteur should have made control experiments," Pasteur replied with some heat, that he had made them. At the next meeting of the Académie he knew more, and had to withdraw his former claim.

With regard to the inoculations from the dog's head, I would ask Dr. Abbott to tell us if he knows of any place, in the Johns Hopkins Hospital or elsewhere, where a systematic attempt has been made to control these inoculation experiments. Has a systematic attempt been made to inoculate rabbits with other materials? and what effects have been produced? and where are the results recorded or published? I know where some such experiments were made, and have seen the animals presenting the symptoms of paralytic rabies. There are a good many things which will give rabbits paralytic symptoms like those of rabies.

One word in closing as to my opinions in regard to hydrophobia; I have acquired them at the cost of much time and labor. I have gathered facts in regard to hydrophobia and rabies from all parts of the world, and I have studied these facts earnestly and honestly, and my views are the result of these endeavors. It may be unreasonable for me to expect those who have not gathered as much evidence as I have on this subject to share my opinion. But no opinions gained so hardly are ever held lightly, and I hold them in the hope that time will show that they are right. There was a time when the belief in witchcraft was supported by all the representatives of learning and religion; but the belief in witchcraft has gone, and I hope to live to see the belief in hydrophobia as a specific inoculable disease go after it.

DR. ABBOTT: In reply to Dr. Dulles's question bearing upon the introduction of non-specific substances beneath the dura mater of rabbits, I

might say that the demonstration of the inability of such materials to produce the characteristic group of symptoms in rabbits is much more frequent than is the production of these symptoms by materials from rabid dogs. By no means are the nervous centres of all suspected dogs that are sent to the laboratory capable of producing this group of symptoms, for comparatively few of them are animals that have died of rabies, and there is usually but little evidence in support of the suspicions with which the animal was surrounded. These cases serve as abundant controls. Moreover, I cannot imagine anyone trained in laboratory methods undertaking work of this kind without controlling the experiments by parallel inoculations with non-specific materials. I do not know to what extent Dr. Dulles has studied this process in the laboratory, but my observations here and in the laboratory of Professor Welch, in Baltimore, leave no doubt that rabies is due to a specific virus which causes the specific group of symptoms, and if this virus is not present the symptoms do not appear.

ROUND-CELLED SARCOMA OF THE ANTERIOR MEDIASTINUM; EXTENSIVE METASTASES, INCLUDING THE BRAIN, BOTH CHOROID COATS, OCULO-MOTOR AND OPTIC NERVES, AND EXTERNAL OCULAR MUSCLES.

By ARTHUR V. MEIGS, M.D.,

AND

G. E. DE SCHWEINITZ, M.D.[1]

[Read February 7, 1894.]

An Italian laborer, aged twenty-one years, was admitted to the Pennsylvania Hospital October 1, 1888. The history was as follows: He was well and strong until June, when he had ague of the tertian type. For forty days before admission he was unable to work, owing to pain in the head and shortness of breath, which for ten days amounted to orthopnœa; his breathing was wheezing. The urine was scanty and passed infrequently. On swallowing he had a burning sensation behind the sternum, and the act provoked coughing, but he did not vomit. He had no appetite and was wakeful at night. There was diplopia for at least ten days before coming to the hospital.

On admission the patient was a fine, strongly developed man. The face was dusky-red and congested, the neck broad and very thick-looking, and its veins and those in the upper part of the chest were full and turgid.

The eyeballs were markedly staring, and there was slight external strabismus of the right eye. The tongue, when protruded, was not deflected to either side.

The arms and hands were cyanotic, but there was no cyanosis of the feet or legs. The dyspnœa was very urgent, and he was entirely unable to lie down.

[1] The case was reported briefly by Dr. T. S. Westcott to the Philadelphia Pathological Society, October 25, 1888, and the report published in the University Medical Magazine, 1889, vol. i.

There was, perhaps, some slight difference in the volume of the two radial pulses at the wrists. The heart-sounds were clear and no murmur was audible. There was dulness on percussion at the lower portions of the lungs posteriorly, and whistling, dry râles were to be heard over the lungs everywhere. There was a roaring sound as if from obstruction of the trachea or bronchi; this was more marked upon the right than upon the left side. He expectorated a good deal of watery, blood-stained fluid.

Death occurred in great agony from suffocation about twenty-four hours after his admission to the hospital.

The *post-mortem* examination revealed the presence of a large fleshy mass lying behind the sternum. This was wedge-shaped, with the base above, and was perhaps three inches wide by five vertically. It lay behind the sternum and over the pericardium, and completely surrounded the blood-vessels and the trachea, except a little of its posterior wall. The trachea was much compressed and pushed or dragged out of the median line.

The innominate artery divided within the mass, and its two branches were narrowed. The carotid, a short distance from its origin, showed destructive erosion so great that the wall was very thin. The left innominate vein was included within the tumor, and it and the carotid and subclavian veins which formed it were all markedly narrowed.

The heart appeared to be of about the normal size, and its valves, cavities, and openings looked natural. Upon its surface the vessels appeared tortuous and thick, and stood out in an unusual way. On cutting across such vessels they appeared to be surrounded with neoplastic material. At the apex there were several spots which were dark-red, resembling ecchymoses. At the base of the heart there was neoplastic material deposited upon the great vessels and on the auricles, and at one place this extended across to the parietal layer of the pericardium, which was ragged-looking.

The pleural sacs both contained bloody serum, the right being tightly distended, and the right lung compressed and carnified. Both lungs showed here and there upon their surfaces spots of neoplastic deposit, and upon the pleural surface of the diaphragm, upon the right side, there were a number of quite large nodules.

The liver was large, weighed four pounds fourteen ounces, and was very full of blood. On section it presented the nutmeg appearance.

The spleen was about five times the natural size and very firm in texture.

The mesenteric glands were greatly enlarged and were sarcomatous. The kidneys presented numerous nodules upon their surfaces, and on section areas of sarcoma were seen scattered through the parenchyma.

The brain itself presented no gross evidence of disease. The oculo-motor nerves were both enlarged, the right one being fully twice as large as the left and presenting a pear-shaped swelling just within the cranial cavity, before its entrance into the sphenoidal fissure.

Sections were prepared, for microscopical examination, of the primary growth which lay in the mediastinum. Of this, sections from three positions were cut, one taken from the body of the mass, and two including the right carotid artery—one of the eroded portion of the vessel, and the other of a portion in which the walls were still whole. Of the heart, sections were cut from two positions, one near the apex, and the other of the septum, on the anterior surface, including an artery in the pericardium. Two sections of the lung were prepared from different positions. Sections were cut of mesenteric glands, of the diaphragm, including a sessile growth upon its pleural surface, of the liver, spleen, and two from different parts of the kidney.

Of the nervous system, sections were prepared of the cerebellum, of both oculo-motor, and of both optic nerves, just within the cranial cavity and before their entrance into the sphenoidal fissures and optic foramina; of the posterior portions of both eyes showing the nerve entrances, and of the optic nerves and surrounding muscles and nerves behind the eyeballs.

The total number, therefore, of different portions of tissue subjected to microscopical examination was twenty-two, and all of them exhibited, to a greater or less extent, sarcomatous infiltration. The infiltrating material was of the commonly described round-celled variety, and in sections stained with carmine the cells seized the pigment with avidity, producing an intensely red color. This seems to be a marked peculiarity of the cells of sarcoma, the color generally being more intense even than that of carcinomatous growths.

The primary growth in the mediastinum, which very probably originated in some remnant of the thymus gland, presented the usual appearances of round-celled sarcoma. The only specially noticeable feature of the growth was the erosion of the carotid artery. The only portion of the walls of the vessel remaining upon the side where the erosion had occurred was the external fibrous coat; the muscular layer and intima had disappeared entirely.

The sections of the heart showed the neoplastic material to lie more abundantly in the adipose tissue upon the surface than anywhere else. It followed the lines of the connective tissue, the rings constituting the boundaries of the fat cells being infiltrated. In places where the amount of deposit was still moderate the centres of the fat cells retained their usual appearance of being empty, but where it was great the cells had pressed inward until the spaces were filled up, and the appearance was one of a solid mass of neoplastic round cells. At the point of junction of the overlying fat with the muscular substance of the heart, the great abundance of the neoplastic material ceased abruptly, as though it had found the invasion of the muscular territory more difficult. The sarcoma cells, however, were plainly to be seen amongst the muscular fibres, especially along the connective-tissue lines, but they were few and sparse as compared with the

vast numbers of them in the fat layer. The invasion by the sarcomatous cells had penetrated very deeply into the muscular tissue, following the lines of the connective tissue and lying between the fibres.

In the lung the sarcomatous infiltration had followed the connective-tissue lines, the pulmonary trabeculæ being enormously thickened and presenting themselves as masses of round cells. The walls of the air cells were also infiltrated in places, and where the growth had fully taken possession of an area, the cells had multiplied so that the air spaces were filled, causing the lung to be quite solid. The nodules upon the pleural surface of the diaphragm were composed of masses of round cells, presenting no unusual features.

In the liver were many areas of varying size infiltrated with round cells, which had stained intensely red. None of these were large, nor had the macroscopical examination revealed the presence of any nodules sufficiently large to be seen with the naked eye. It seems fair, however, in view of the fact that sarcomatous infiltration had occurred so extensively into so many tissues and organs, to assert that these small infiltrations into the liver were but an early stage of the same malignant disease which had progressed so much further in other places.

The spleen and mesenteric glands were sarcomatous, as was easily seen from their gross appearance, but as their natural histological appearances so closely resemble those of a round-celled sarcoma, the only specially noticeable characteristic of them was the intense red staining of their cells.

The kidneys presented numerous nodules, both upon the surface and more deeply seated within their substance. These, when examined microscopically, were seen to be sarcomatous, the neoplastic cells lying between the bloodvessels and renal tubules in the connective tissue, except where the deposit had become extensive, in which case the renal tissue was overwhelmed, and the appearance was simply that of a mass of the characteristic round cells. In one place there was found a layer, somewhat less than a thirty-second of an inch in depth, of sarcomatous deposit upon the surface of the kidney. The neoplasm included the capsule, and the separation of the diseased from the healthy renal tissue beneath it was very sharply marked. Hemorrhage into this neoplastic material directly beneath the capsule had occurred.

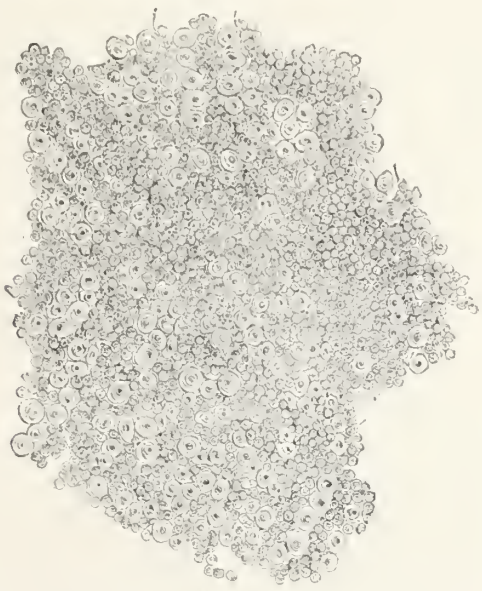
The appearances of the sections of the cerebellum were not easy to interpret. The portions of pia mater included were infiltrated, especially around the bloodvessels, with cells precisely similar in appearance to those which in other places were beyond doubt sarcomatous. In view of this fact it seems fair to assert that the disease had invaded the pia mater, and this is the more just and reasonable, as a precisely parallel condition existed in connection with the eyes, as may be seen from the following description:

Left oculo-motor nerve. The individual nerve fibres are of normal appearance, axis-cylinder and medullary substance presenting their natural charac-

teristies. Here the normal constitution ceases, and the nerve bundles, and in many places the individual fibres, are separated by collections of round sarcoma cells, which have closely followed the connective-tissue septa and the bloodvessels supported by them. In other words, instead of the ordinary membranous investment of the fasciculi, this has been changed into one composed of the cellular elements of the sarcoma.

In the *right oculo-motor nerve* precisely the same process is repeated, only the infiltration of the perineurium and endoneurium is so extensive that the appearance is that of a section of ordinary round-celled sarcoma, through

FIG. 1.



Section of right oculo-motor nerve showing extensive sarcoma-infiltration.

which are interspersed here and there single, normally constituted nerve fibres. (Fig. 1.)

Portion of the posterior half of the right eyeball and 9 mm. of the optic nerve in longitudinal section. Beginning from the extra-ocular end of the optic nerve and passing to the lamina cribrosa, there is no marked change in the nerve bundles and no increase in the nuclei. The trabeculæ of connective tissue are more developed than in the normal nerve, but are not infiltrated with sarcoma cells. Toward the extra-ocular end of the nerve are several capillary vessels, around which are clustered numerous round cells. The lumen of the central arteries contains a few blood cells, and in addition small collections of larger, deeply stained lymphoid cells—a condition which re-

peats itself more decidedly in several adjoining vessels or branches. The outer sheath is thickened, but not infiltrated; the inner sheath is unchanged. The intervaginal space contains numerous darkly stained round cells gathered in the meshes of loose fibres. The nerve entrance is free from changes, evidences of neuritis being entirely absent.

The *retina* is badly broken and misplaced, rendering an accurate study of its elements difficult, which, save for some proliferation of the internal nuclear layer and distinct thickening of the fibre layer, are free from pathological changes. The pigmented epithelium appears only in fragments clinging to the choroid coat.

The *choroid* to the left of the nerve entrance, so far as the lamina vitrea and chorio-capillaris are concerned, presents no abnormalities. The blood-vessels of the stroma are well filled with corpuscles and some darkly stained cells. Here and there the leucocytes of this region are gathered together in small collections. The pigment cells, very dark, appear in characteristic variety of shapes.

To the right of the optic nerve the choroid is detached from the sclera, is thicker than on the opposite side, and, in addition to the appearances just

FIG. 2.

Section of left choroid, showing large bloodvessels, pigmented cells, and secondary sarcoma-deposit.

described, presents in several spots in close relationship with vessels in the stroma considerable collections of round, darkly-stained cells, which, in general character and grouping, undoubtedly belong to the sarcomatous infiltration.

The *sclera* is free from abnormal appearances, but in the branches of the ciliary nerves passing through it there are small round cells suggestive of those which have infiltrated the oculo-motor to such a great extent.

Posterior half of the left eyeball and 8 mm. of the optic nerve in longitudinal section. So far as the optic nerve, optic-nerve entrance, retina, one-half (left) of choroid and sclera are concerned, the description just given applies so nearly that it need not be repeated. The right half of the choroid remains *in situ* for 3 mm. from the optic-nerve entrance. Then it becomes detached from the sclera and is greatly thickened. The branching, pigmented cells are massed together on the outer part, and also surround the well-filled vessels of the stroma, between which are gathered areas of round sarcoma cells, interspersed with pigment granules and dark, spindle-shaped cells. The chorio-capillaris can be traced unbroken throughout the section, but is indistinct directly over the thickest part of the affected choroidal tissue. The sarcoma cells are dispersed through the layer of choroidal stroma containing large bloodvessels, but are especially massed in several localized areas. Some of the larger veins contain darkly stained cells analogous in their appearance to those which lie outside of the vessel walls. (Fig. 2.) This area of infiltration begins 3 mm. from the nerve entrance, and is 1 mm. in diameter at its thickest portion.

Transverse section of the contents of the left orbit about 14 mm. anterior to the foramen opticum.[1] The relation of the parts (muscles, vessels, nerves, etc.) one to the other is undisturbed.

Muscles. The external, internal, superior rectus, and levator palpebræ superioris are normal in appearance; the inferior oblique is not included in the section. The inferior rectus contains a patch, constituting about one-third of the section, situated in the upper part, which presents an exquisite picture of infiltration with round sarcoma cells. The connective-tissue investments of the individual fibres and of the fasciculi are literally replaced with the cells of the new growth; while the sheath of areolar tissue covering the entire muscle in the affected area is crowded with the elements of the neoplasm, which extend into the surrounding fibro-fatty tissue of the orbit. For the most part the diseased area is sharply separated from the portions of the muscle which remain unaffected, although in places small trains of cells proceed a short distance into neighboring areas along the internal perimysium.

The section of the superior oblique is more freely supplied with darkly stained nuclei and corpuscles than those of the other muscles, and in one or two spots, in the neighborhood of small bloodvessels, are collections of larger lymphoid cells, probably representing small foci of sarcomatous infiltration. (Figs. 3 and 4.)

[1] This section corresponds almost exactly with Table VI. of Lange's Topographische Anatomie des menschlichen Orbital-Inhalts, and has been studied with the aid of this diagram.

Optic nerve. The nerve itself is normal, but in the intervaginal space, clinging closely to the inner sheath, are many small round cells, while in the fibro-fatty tissue, between the nerve sheaths and the inferior rectus muscles, are numerous collections of sarcoma cells, particularly immediately in relation with the cross sections of the ciliary arteries.

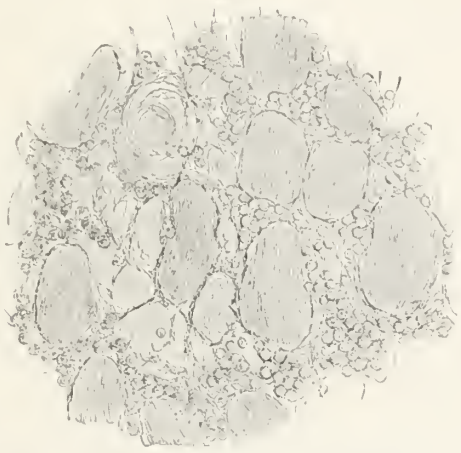
FIG. 3

Transverse section of contents of left orbit 14 mm. anterior to foramen opticum, showing, A, patch of sarcoma in inferior rectus; B, C, D, E, external, internal, and superior rectus, and levator, unaffected; F, superior oblique; sarcoma cells lie between optic nerve and inferior rectus.

Other nerves. The ciliary nerves, naso-ciliary nerve, branches of the oculo-motor, supra-orbital, and a branch of the trochlear have been identified. In none of them are the appearances repeated which have been described with the main stem of the oculo-motor. Of the nerves just named the supra-orbital is more richly supplied with corpuscles and nuclei than the others, but there are no sarcoma cells between the nerve fibrils. The branches of

the nerves supplying the inferior rectus, contained within the body of the muscle, although themselves not infiltrated, are liberally surrounded with sarcoma cells, which infiltrate their external connective-tissue investments.

Fig. 1.



Section of inferior rectus showing sarcoma cells between muscle-fibres.

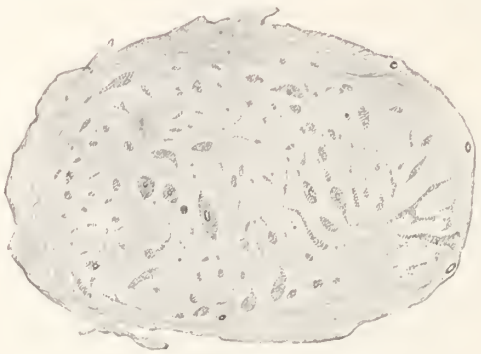
Bloodvessels. The following bloodvessels are evident in the section: the supra-orbital, ophthalmic, and ciliary arteries and branches of the ophthalmic veins. The coats of these vessels are free from infiltration, except in the case of the ciliary branches lying between the optic nerve and the patch of infiltration in the inferior rectus, and even in these the sarcoma cells lie rather in the loose fibro-fatty tissue adjacent to the vessels than in coats of the artery itself, although in some instances small foci may be seen in the adventitious tunic. In the small vessels supplying the inferior rectus muscle this character of infiltration (*i. e.*, within the adventitious coat) is more conspicuous.

Transverse section of a portion of the contents of the right orbit. As the contents were fragmentary, the relation of the various constituents to each other, and consequently their identification, is imperfect.

Muscles. The following muscles appear to be included: superior rectus, levator palpebræ superioris, a fragment of the superior oblique, and the internal (or inferior) rectus. These structures have escaped the sarcomatous infiltration, with the exception of the last-named muscle, which contains two patches of diseased tissue exactly similar to the one described in the inferior rectus of the left orbit. The branches of the oculo-motor nerve supplying this muscle are not only surrounded by sarcoma cells, but the individual fibres have been separated by the elements of the new growth precisely as the same process has occurred in the main stems of both third nerves.

The optic nerve is normal in appearance, but in the intervaginal space the collections of round cells are more evident than in the corresponding situation on the opposite side.

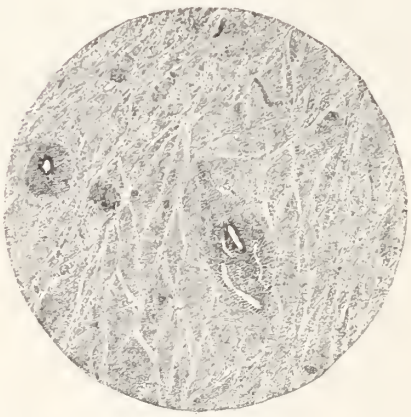
FIG. 5.



Transverse section of optic nerve just within skull, showing general plan of the sarcoma-infiltration.

The bloodvessels, except the small branches which supply the muscle in the area of infiltration, are free from sarcomatous deposits.

FIG. 6.



Portion of Fig. 5 more highly magnified, showing arrangement of sarcoma cells surrounding the bloodvessels.

Cross-sections of the optic nerves just within the skull. The dural sheaths are not visible in the section; the inner (pial) sheath is densely infiltrated with sarcoma cells, which, passing inward from this situation along the trabeculæ, infiltrate those in and within the margins of the nerves. In

general, the nervous tissue appears normal, and there is no undue development of nuclei. Each capillary and arteriole, however, is surrounded with a considerable area of neoplastic cells, which completely replace the adventitious coat and peri-vascular sheaths of the larger vessels, and entirely fill the spaces surrounding the smaller ones. Within the lumen of many of the vessels are cells exactly the counterpart of those which surround them. (Figs. 5 and 6.)

The case seems worthy of being described and recorded, as sarcoma of the mediastinum is by no means common, and as the metastatic growths were more fully examined and studied with the microscope than can or has been done in most of the cases recorded in the journals, but especially on account of the condition of the eyes and their appendages. As has already been said in connection with the description of the macroscopic conditions, at the post-mortem examination nothing abnormal was seen except the difference in size of the two oculomotor nerves. It would probably have escaped notice that the left, the smaller of the two nerves, was much larger than usual, had it not been for the fact that the right was still larger. The difference in size having been observed, of course further investigation was made to discover the cause.

Since seeing this case, now four years ago, one of us (Dr. Meigs) has seen another, which was in many respects parallel:

A man, aged forty-three years, who had always been healthy, was for a month annoyed by slight cough; but it was so slight that it had not prevented his starting on a long pleasure journey. Two weeks later he found he had shortness of breath, which soon became so severe that he was advised to come home. When seen he was dull of intellect and his mind disposed to wander; he had dyspnœa, and the face was much congested and swollen. There was marked nystagmus, and the eyeballs seemed prominent. In the supra-clavicular fossa upon the right side was a tumor. This was not very hard and seemed to be pressing upward from the chest cavity, so that it almost entirely filled the fossa. It did not pulsate. Examination of the heart brought to light no evidence of disease, as the sounds were soft and clear, but the action was rather feeble. There were whistling and cooing râles to be heard over the lungs, but no signs of consolidation. The appearance of the lower extremities was in marked contrast to that of the head. The patient was a large, stout man, and the swollen head and bulging eyes hardly looked as if they could be a part of the same individual as the legs,

which were emaciated and shrunken. The history was that the tumor had existed only two weeks. This, taken in consideration with the fact that there had been severe dyspnœa for the same length of time, indicated strongly the presence of a rapidly growing intra-thoracic mass. Such a growth could only be a sarcoma, and the opinion was therefore expressed that the disease was mediastinal sarcoma.

It seemed almost certain, too, in the light of the other case that has been described, that the staring of the eyeballs and nystagmus must be due to an invasion of some part of the ocular apparatus by metastatic sarcoma. Within three or four days a tumor showed itself in the other supra-clavicular space, the dyspnœa increased terribly, and he died. A post-mortem examination was made, and a sarcomatous tumor weighing one and a quarter pounds was found occupying the mediastinum. The growth surrounded the trachea, aorta, and other large bloodvessels, and there were secondary deposits ranging in size from that of a bean to a chestnut upon the diaphragm and posterior wall of the thorax. The post-mortem examination does not appear to have been very thoroughly made, for there is no mention of metastases to any of the organs, though such almost certainly must have existed, nor was any investigation of the brain or eyes made. It is much to be deplored that sections from the eyes and their appendages could not have been thoroughly examined with the microscope, for the disease had almost certainly invaded them. That which was observed in this case, imperfect as the investigation was, when taken in connection with the one more fully described, makes it desirable that metastases to the ocular apparatus in cases of mediastinal sarcoma should be sought for. It may be, too, that it occurs in cases of sarcoma arising in other situations than the mediastinum. The presence of the sarcomatous deposits in the ocular apparatus might readily be overlooked, for there are hardly any gross appearances to be seen. It is reasonable to suppose that there was more sarcomatous deposit somewhere in the brain than that in the pia mater of the cerebellum which has been described, and which could only have been discovered by careful microscopical examination.

As already indicated, a large measure of interest centres in the distribution of the sarcoma cells within the eyes, optic nerves, oculo-motor (third) nerves, and the orbital muscles. Both third nerves are extensively infiltrated throughout their length, especially upon the right side. In the branches of the nerves in the left orbit no exactly similar appearance was found, except in those supplying the inferior rectus, which, while not themselves infiltrated, are densely surrounded by cells. Again, in the right orbit, of the muscles apparently identified, a portion of the internal (or inferior) rectus is contaminated, as also are the nerves supplying it, not merely in the form of surrounding areas of cells, but also by a separation of the individual fibres with the elements of the new growth, exactly as this occurs in the parent stem. Therefore, if the appearances seen in the left superior oblique are not accepted as certainly sarcomatous, we may infer that one pathway of the morbid process was along the oculo-motor nerves, and through them to two, possibly three, of the muscles which they supply.

The optic nerves within the skull are extensively diseased, but the process of infiltration grows less marked as they approach the eyes themselves. Indeed, even in the cross-section in the orbit scarcely any diseased tissue is evident, while in the portions of these nerves examined in connection with the posterior halves of the eyeballs, save for some slight cellular infiltration around the capillary vessels and in the intervaginal space, evidence of sarcoma is lacking. Therefore it may be inferred that a second pathway of the morbid process was *via* the optic nerves, and a striking and interesting feature of the case at once becomes apparent, viz.: that the disease was more advanced and extensive in degree within the skull than in the orbital and ocular termination of the third and second nerves. In other words, this indicates that the disease travelled along these pathways from the brain forward toward the eyes.

The deposits of sarcoma cells in each choroid, but particularly in that of the left eye, give this case importance in the

literature pertaining to sarcoma of the uveal tract. The fact that these deposits are limited to a certain area of the choroid, that they are surrounded by comparatively healthy tissue not contiguous with infiltrations elsewhere located, and that the bloodvessels within them and in their immediate vicinity contain cells apparently of the same character, affords evidence that they should be regarded as metastatic nodules, probably of embolic origin.

Fuchs, in his admirable monograph, *Das Sarcom des Uveal Tractus*, Wien, 1882, having analyzed the entire literature to date, states that metastatic choroidal sarcoma is unknown, and quotes Virchow's well-known sentence: "Those organs which exhibit a great tendency to protopathic tumor formation present a very slight inclination to metastatic deposits." Fuchs refers to, but rejects, Broemser's case[1] of supposed metastasis of a melanotic growth of the cheek to the choroid, for the excellent reason that no microscopic evidence is at hand that either the ocular or the facial growth was sarcomatous, and reiterates his belief that up to the date of his writing sarcoma metastasis to the choroid had not been observed.

Pflüger,[2] of Bern, contributes a case of sarcoma in a young woman, aged thirty years, which developed from a congenital nævus in the region of the right parotid gland, and which was followed by secondary sarcoma of the glands, with metastasis into the skin of the back and head, into the right choroid, and probably into the cerebrum. Three years and a few months elapsed between the period when the congenital pigment-patch began to enlarge and the death of the patient. Autopsy was refused, and the facts reported are based upon clinical observation alone. Ophthalmoscopic examination revealed a tumor occupying the medial half of the eyeground. Pflüger refers to another case of intra-ocular metastatic sarcoma reported by Schiess-Gemuseus,[3] the primary growth being a sarcoma originating from a congenital nævus in the parotid region.

[1] Ueber einen Fall v. secundär Melanom. d. Choroid. Diss. Inaug., Berlin, 1870.

[2] Archives of Ophthalmology, vol. xiv. p. 185.

[3] Graefe's Archiv, Bd. xxv., Abth. 2.

Ophthalmoscopic examination was impossible, owing to opacities in the media, but the microscope showed that the secondary growth had originated from the optic papilla. Hence the case is not properly classed with choroidal growths.

In the case forming the basis of this paper, unfortunately ophthalmoscopic examination is wanting, but the anatomical studies are sufficiently detailed to demonstrate secondary choroidal sarcoma. It is scarcely conceivable that the choroidal growth under these circumstances could have been the primary one, although, as is well known in a few instances, extremely small and totally unsuspected growths in this situation have been followed by very extensive metastasis; in one case quoted by Fuchs, a melanotic mass was found in the heart secondary to a small sarcoma in a shrunken eyeball, which had existed in this condition for more than twenty years.

Independently of the fact that the sarcomatous deposits in the eye were confined within the scleral walls, and were both clinically and microscopically of the nature of secondary growths, the mediastinum rarely suffers from this form of tumor, save as a primary growth. Among ninety-eight cases reported by various authors and collected by H. A. Hare,[1] but five were secondary and thirty-one were primary, the remaining number having no distinct reference in regard to this point. The interesting fact has been noted that so far as the optic nerves, third nerves, and orbital contents are concerned, the microscopic evidence is that the disease travelled from the brain forward toward the eyes along these pathways, but the localized character of the choroidal neoplasms and their failure to be in any demonstrable connection with other sarcomatous areas, seems definitely to indicate that they should be regarded as embolic deposits.

[1] The Pathology, Clinical History, and Diagnosis of Affections of the Mediastinum. Fothergillian Prize Essay. Philadelphia, 1889.

SUCCESSFUL SIMULTANEOUS TRIPLE AMPUTATION.

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[Read February 7, 1894.]

L. J., aged twenty-one years, was admitted to the University Hospital on the night of October 20, 1893, having a short time before been run over by a train on the Philadelphia, Wilmington, and Baltimore Railroad. Upon examination after his admission, it was found that he had sustained a crush of the right leg extending to the knee-joint, the left leg was crushed and torn off in its middle third, and the right hand and lower part of the right forearm were completely crushed. At the time of his admission there was little bleeding from the injured parts, although it was stated that he had lost a large amount of blood before he was brought to the hospital. He exhibited marked symptoms of shock, his surface being cold and his temperature a little over 96° F. Esmarch's elastic straps were wrapped around the lacerated tissues of the limbs to control any bleeding which was present, and the patient placed upon the operating-table and surrounded by hot-water cans and covered with blankets; his clothing was only removed so far as to expose freely the injured parts. He was given one-thirtieth of a grain of strychnia hypodermatically, and this dose was repeated in half an hour. When I saw him for Prof. Ashhurst, into whose service he came, about an hour after his admission to the hospital, I found that he was reacting from the shock, his temperature was 98° F., and although his pulse was rapid it had some volume. Dr. Girvin, the resident surgeon in charge of the patient, stated that his condition had markedly improved since admission. Under the circumstances I considered that an attempt to remove the crushed limbs was justifiable. The patient was again given one-thirtieth of a grain of strychnia hypodermatically and carefully etherized, and Esmarch straps were applied to each of the injured limbs some distance above the proposed points of amputation. The right limb was first prepared for operation by washing with soap and water and bichloride solution, and I amputated this limb just above the knee, making antero-posterior flaps; the vessels were secured by ligatures. While the first limb was being removed the other limbs were prepared

for operation by my assistants, so that little time was lost, and I amputated next the left leg a short distance below the knee-joint, making lateral flaps, and the right forearm in its middle third. Having secured the vessels, the stumps were closed and dressed with a bichloride-gauze dressing.

The patient lost very little blood during the operation, and his condition after its completion seemed to me to be almost as good as before it was begun.

He was given strychnia, one-thirtieth of a grain, several times during the operation, receiving in all from the time of his admission five injections hypodermatically, of one-thirtieth of a grain each. The time consumed in amputating the limbs and dressing the stumps was about an hour and twenty minutes. The patient was placed on his bed after the operation and kept surrounded with hot-water cans for twelve hours. Liquid diet and about four ounces of whiskey in twenty-four hours, and five grains of ammonium carbonate every two hours, were given. His recovery was uninterrupted, and

I have the pleasure of showing him to you this evening with his stumps perfectly healed.

A few cases of successful simultaneous triple amputations have been reported by Prof. Ashhurst, and Drs. Jackson, Wallace, Lowman, and others, but the majority of patients who have suffered from injuries requiring this procedure perish promptly from hemorrhage and shock, or are in such a condition when they come under the surgeon's care that no operative treatment can be instituted. Although the mortality following multiple amputations is high, there is no reason why recoveries should not be more numerous under modern methods of wound treatment if the element of shock could be eliminated. When, however, a case comes under the care of the surgeon, whose condition is such that it seems justifiable to amputate a number of limbs at the same time, I think there are certain details which should be carefully carried out.

In the first place, such cases usually present a condition of marked shock; this is to be overcome by the application of external heat, and by the use of strychnia hypodermatically, the value of this drug as a heart stimulant in such cases being unquestionable. Stimulants administered by the mouth are usually vomited promptly, and have little effect. Strong black coffee, as hot as the patient can comfortably swallow, is often retained, and in my experience is one of the most useful stimulants that can be taken by the mouth.

The practice of stripping the patient immediately and putting clean clothing upon him, by which means his surface is exposed and chilled, is to be condemned. If the clothing is dry I do not have it removed, but cut away such portions as are necessary to get a free exposure of the injured parts, and often allow the clothing which he wears at the time of the accident to remain upon him until he has thoroughly reacted, often for some days after the operation. As regards the method of amputating, some surgeons consider it advisable to have the limbs amputated at the same time by different operators, so that the patient need be under the influence of the anæsthetic for the shortest possible time.

I fail to see the advantage of this method of procedure, for

it seems to me that the shock of the operations when thus done synchronously must be greater than when the parts are removed one at a time, as was done in the case I have shown.

I make it a rule to amputate first the limb which has received the greatest injury, and whose removal would likely be accompanied by the most shock, and, if the patient's condition is not markedly affected by its removal, I then operate upon the second or third, as the case may be. If, however, I find the patient's condition becomes decidedly worse after the first limb has been removed, and that he would not possibly survive the removal of the other member or members at the same time, I desist from further operative interference, and wrap the other injured parts in an antiseptic gauze dressing and wait until he reacts, which may be in twelve or twenty-four hours; and when reaction has been established, I undertake the further amputation or amputations that may be required. I can possibly illustrate this better by reporting a case in which I adopted this procedure. During my service at the Presbyterian Hospital, in the summer of 1892, there was admittted a switchman who had been thrown from the top of a car and run over, sustaining a crush of both legs below the knee, and an extensive compound comminuted fracture of the shaft of the left femur. As the patient had reacted when I saw him, I amputated his left leg and removed a number of large fragments from the fractured femur, and drained and closed the wounds. Upon examining the patient, before operating upon the other leg, I found that his condition was so unfavorable that I did not think he would survive the removal of the second limb; his temperature, which had been nearly normal before the removal of the first limb, had fallen to 96° F., his pulse was very feeble, and his skin was covered with a cold sweat. I therefore had the other injured limb wrapped in towels which had been wrung out of bichloride solution, and the patient was treated actively to bring about reaction from the shock. He reacted fully, and when I saw him about eight hours afterward his temperature was 99° F., his skin was dry, and he had a good pulse. He was then etherized and I amputated the second limb; he made an uneventful recovery.

A FURTHER COMMUNICATION ON THE RESULTS OF A BACTERIOLOGICAL EXAMINATION OF THE PIPETTES AND COLLYRIA TAKEN FROM A TREATMENT CASE USED IN OPHTHALMIC PRACTICE, WITH THE EFFECTS OF INOCULATIONS.

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[Read March 7th, 1894.]

At the meeting of the College of Physicians, April 3, 1893, we presented a preliminary communication upon the subject contained in the title,[1] and the results which were reached are summarized in the following tables which are here reproduced.

The examination had not gone sufficiently far at that time to plate the cultures and separate the organisms which were present in the lotions and pipettes. The termination of this research is as follows:

The cultures given, both by the cocaine pipette and cocaine solution, were found to contain the *micrococcus aquatilis*, the *bacillus liquefaciens*, and the *proteus vulgaris*. In addition, the *bacillus of Vignal* was detected, which must have been an accidental and unusual contamination.

[1] In this paper the previous work in this line of research was reviewed.

Pipettes.

Unused pipette.	No growth.		
Cocaine pipette.	Growth on potato, agar-agar, and beef-broth.	Same germs as those found in cocaine solution; inoculation caused purulent irido-choroiditis.	Rubbing abraded cornea with culture produced moderate ciliary injection and slight clouding of cornea.
Fluorescine pipette.	Growth on potato.	Inoculation caused slight iritis.	
Atropine pipette.	No growth.	Pipette had been frequently cleansed with sublimate lotion.	
Pyoktanin pipette.	No growth.		
Homatropine pipette.	No growth.		
Eserine pipette.	Growth on all three culture media.	Slight iritis caused by inoculation, which speedily disappeared.	
Boric acid pipette.	No growth.	This pipette had often been cleansed with sublimate after use.	

Fluids.

Boric acid lotion.	Active growth on all three culture media.	Inoculation into anterior chamber produced purulent irido-choroiditis.	Rubbing abraded cornea with culture produced moderate ciliary injection and slight clouding of cornea.
Atropine lotion.	No growth at first; 1 month later active growth.	The growth, a fungus, was not used in inoculation, as it was of same nature as that obtained from the fluorescine dropper.	
Homatropine lotion.	Growth on all three culture media.	Inoculation produced slight iritis, which disappeared in fifteen days.	
Cocaine lotion.	No growth at first; 2 months later active growth on all three culture media.	Inoculation produced purulent irido-choroiditis.	Rubbing abraded cornea with culture produced moderate ciliary injection and slight clouding of cornea.
Fluorescine lotion.	Fungus growth on agar-agar.	Not used in inoculation; growth same as from dropper.	
Bichloride lotion.	No growth.		
Nitrate of silver lotion.	No growth.		
Eserine lotion.	No growth.		

The boric acid solution contained the same germs as those found in cocaine, with the exception of the bacillus of Vignal.

The atropine lotion, which, when first examined, showed no growth but later a fungus, contained two months after the first examination the *micrococcus prodigiosus* and the *bacillus liquefaciens.*

The homatropine lotion contained the *bacillus liquefaciens* and a fungus.

At the first examination the eserine pipette produced a growth on three different culture media, but the solution of the drug appeared sterile. Two months later the same solution, although tightly corked, showed a marked fungus growth (aspergillus glaucus). The *bacillus liquefaciens* and *bacillus implexus* were also present.

Inoculations into the eyes of rabbits with the individual cultures were made in the same way as in our previous experiments, namely, one-tenth of a cubic centimetre of an emulsion of the surface growth upon agar in the water of condensation, was injected, with all proper precautions, into the anterior chamber by means of a syringe. The daily appearances of the rabbits' eyes were exactly the same as those detailed in the first research, and need not be repeated.

The injurious effects of the inoculations from the cocaine and boric acid solutions, which were the same in both cases, were due to the *bacillus proteus vulgaris.* The *bacillus liquefaciens* produced an iritis, or more properly, a hyperæmia of the iris, which disappeared in a week.

The *micrococcus prodigiosus* from the atropine solution produced the same effect, namely, a purulent iritis, while the *bacillus liquefaciens* caused a moderate iritis or hyperæmia.

An inoculation of the culture of the *bacillus implexus* obtained from the eserine solution caused a purulent inflammation, while the *bacillus liquefaciens* obtained from the same source provoked only a moderate hyperæmia of the iris.

As has several times been shown by observers, and as we have chemically proven, an eserine solution which contains the fungus growth no longer responds to the characteristic tests

for eserine. Francke has found that solutions of atropine and eserine, sterile at first, after standing for some time become contaminated, even if the bottles have been carefully corked. The germs appear to get in at the sides of the stoppers. This, it will be remembered, was the experience in our experiments.

It becomes evident, then, that the following organisms are liable, sooner or later, to infest solutions of the alkaloids used in ophthalmic practice, namely, the *micrococcus aquatilis*, the *bacillus liquefaciens*, the *proteus vulgaris*, the *micrococcus prodigiosus*, the *bacillus implexus* (perhaps the *bacillus of Vignal*), and various fungi, particularly the *aspergillus glaucus*. Of these organisms the *proteus vulgaris*, the *micrococcus prodigiosus*, and the *bacillus implexus* introduced into the anterior chamber are capable of producing a purulent inflammation of the iris, cornea, and deeper coats of the eye. Exactly similar inoculations with the *bacillus liquefaciens* resulted in a hyperæmia of the iris, possibly a slight iritis, which disappeared in a week. The *micrococcus aquatilis* had no effect.

The growth of fungus in these solutions appears to have little deleterious effect upon the eye, but, as already noted, has the power, with eserine, at least, of changing its chemical composition. As shown in the first research, these organisms when inoculated upon an abraded cornea, the anterior chamber not being open, produce a moderate amount of ciliary injection and clouding of the corneal structure, but did not in any of our experiments call into existence a purulent inflammation of the eye.

It is unnecessary to repeat the methods of properly sterilizing alkaloidal solutions. These were summarized in the first paper, and have been published many times previously. Perhaps the most ingenious device for this purpose is the one introduced by Dr. Strosehein, of Würzburg.[1]

[1] It may be stated that the experiments were performed in the laboratory of Dr. E. A. de Schweinitz, and that the separation of the micro-organisms and the inoculations are entirely his work. We are indebted to Dr. William M. Gray for preparing the slides which showed the lesions of an extensive purulent irido-choroiditis, both in the present and previous research.

HYSTERECTOMY FOR OTHER CONDITIONS THAN FIBROID AND MALIGNANT TUMORS.

By CHARLES B. PENROSE, M.D.

[Read March 7th, 1894.]

Until very recently the operation of hysterectomy was confined practically to two conditions, malignant disease and fibroma. The improved technique and the admirable results obtained in the modern operation for these conditions have led to its extension to other diseases of the uterus and the appendages. The most radical proposition is: to remove the uterus in every woman in whom it is necessary to remove the uterine appendages. A second less radical proposition may be thus stated:

In any case of pelvic disease in women when there is necessary for cure an operation on the appendages which renders her sterile, hysterectomy is a valuable addition to the operation under the following circumstances: when the uterus is diseased, when it is likely to become so, when its removal will facilitate the operation.

I have been following this latter plan since June, 1893, and wish to report my results and conclusions to the Fellows of the College with the object of obtaining their views upon this new plan of treatment.

The number of cases is not large, but represents a variety of conditions. In a series of seventy-five cœliotomies on women for different forms of pelvic disease exclusive of fibroid and malignant tumors, I have found it of distinct advantage to

remove the uterus in fifteen cases, which may be grouped under the following headings:

1. Cases of double salpingitis—generally pyosalpinx—the disease extending into the uterine cornua in the form of a hard cheesy nodule, or an abscess in the uterine tissue; large uterus, chronic metritis and endometritis; profuse irritating vaginal discharge, probably gonorrhœal.

2. Cases of salpingitis and ovaritis; flexed or displaced uterus—with or without adhesions, endometritis.

3. Salpingitis of which tuberculosis is the probable cause.

4. Cases of ruptured tubal pregnancy, of pelvic abscess, or of any accumulation in the pelvis where the uterus forms part of the wall surrounding the accumulation. The removal of the uterus facilitates the operation and perfects hæmostasis.

It must be remembered that in all these cases the uterus was removed as an addition to an operation which rendered the woman sterile. I think that we can consider that the removal of the Fallopian tubes renders the woman sterile except in very rare instances.

The gonorrhœal cases form a large class representing the most frequent form of salpingitis; and in this disease I think that hysterectomy is a valuable addition to the old operation of simple salpingectomy. In several of the gonorrhœal cases my determination to perform hysterectomy was based on the fact that gonococci had been found in the discharge from the os uteri. In others the history of the case, and the presence of gonorrhœal lesions as inflammation of the vulvo-vaginal glands or their ducts, or urethritis, indicated the gonorrhœal origin of the disease; and finally in other cases gonococci were sought for by an assistant in the tubal contents during the operation.

In cases of gonorrhœal pyosalpinx the old operation of removal of the tubes relieves the woman from the danger of rupture into the peritoneum. But it does not cure her. The disease continues in the endometrium and in the deeper structures of the uterine wall, often extending from the tubes into the uterine cornua. Careful operators have been in the habit of

removing, by a wedge-shaped amputation, the proximal end of the Fallopian tube in these cases. This operation, however, requires almost the same length of time as hysterectomy, and is not followed by perfect hæmostasis.

The operation of hysterectomy, however, cures the woman of the leucorrhœa, which is often the symptom of which she complains the most.

I have tried, by all the means devised, to cure gonorrhœal endometritis, with but very unsatisfactory results. I have never cured it in a case where pyosalpinx had coexisted.

If in these cases we leave the uterus, the woman continues to have an irritating infectious discharge, and in this condition she is not considered cured by herself or her husband. In posterior displacement with salpingitis and adhesions to the fundus uteri, removal of the uterus gives more promise of cure than simple removal of the tubes.

It is a mistake to suppose that the posterior displacement will remain corrected by the tension of the broad ligaments following a simple salpingectomy. In one case in my series the tubes and ovaries had been removed some years before my operation, and yet the uterus was in a position of extreme retroversion, and the pressure of the fundus upon the rectum caused such pain that for some months before the operation every movement of the bowels had been preceded by a hypodermatic injection of morphine. Hysterectomy stopped the pains and the morphine habit.

In tubercular disease of the tubes I think that hysterectomy should always be performed. The investigations of Williams, *Johns Hopkins Hospital Reports*, vol. iii., show that in this disease the uterus and endometrium are affected in from 40 to 60 per cent. of the cases. Unfortunately, it is at present impossible to determine the tubercular character of many cases of salpingitis by the gross appearance, at the time of operation, and we are unable to determine the full extension of the disease.

In a case of salpingectomy done last November, the microscope showed tubercular infiltration in the tubes extending to

the plane of section. Had I known this at the time of operation I should have removed the uterus.

In one case of suspected tuberculosis I curetted the uterus and examined the scrapings before the operation, and then performed hysterectomy on account of the probable tubercular character of the endometritis.

The fourth class of cases, in which the operation is facilitated by the addition of hysterectomy, is illustrated by a case of ruptured tubal pregnancy. The woman had a ruptured tubal pregnancy on the left side. The pelvis was filled with old blood-clot, which was walled in by adherent intestines above, and by the uterus and broad ligaments in front. The abdominal ostium of the right tube was closed, and the tubes and ovary were bound down by adhesions. In this case the removal only of the blood-clot and as much of the sac as practicable would have left behind a large bleeding uterus destitute on two-thirds of its surface of any peritoneal covering, and functionally useless when the tubes and ovaries were gone on each side. Its removal, however, enabled me to secure quick and complete hæmostasis and to avoid an elaborate drainage of the pelvis.

I know of no way in which the removal of what has been called the emasculated uterus injures the woman. The results in large numbers of hysterectomies for cancer and fibromas show that, from the mechanical and functional points of view, the remaining pelvic contents are uninjured.

The uterus does not seem to be, like the ovary, necessary in any way for the maintenance of womanly traits. I think that in cases of salpingitis requiring treatment which renders the woman sterile, the removal of the uterus along with the tubes does her less harm from a psychological standpoint than the common operation of removing the ovaries with the tubes. I think that the ovaries or parts of them should be left in all cases where practicable—especially in young women.

An objection urged against this form of hysterectomy is the increased danger of the operation. It is undoubtedly true that in some cases the prolonged time of operation, even if only

ten or fifteen minutes, may be of serious harm to the woman. In such cases I consider that the operation is improper. The determination to remove the uterus in the class of cases under consideration should depend upon the condition of the woman when she is on the operating-table. If her condition is such that she can endure the increased operation, then I think that hysterectomy is a valuable addition to our treatment in all cases of tubal and ovarian diseases requiring an operation which renders the woman sterile, provided that the uterus is itself diseased to such an extent that it will not readily yield to local treatment, or is so implicated in the pathological condition in the pelvis that its removal facilitates the performance of a complete operation.

The statistics with which I am familiar show that the removal of the uterus under these circumstances is attended with as small mortality as the operation of salpingectomy. All the cases in my own experience have recovered.

DISCUSSION.

Dr. J. M. Baldy: The paper brings the subject under consideration practically down to hysterectomy for pelvic inflammatory disease. Where there is tuberculosis I think that there would be no objection to the proposition that the uterus should be removed together with the appendages. In fully one-half these cases the uterus is involved.

With regard to removal of the uterus in pelvic inflammatory conditions there has been a great deal of discussion, and there is considerable difference of opinion as to which cases should be operated on and which should not. The dictum has gone forth in New York that the removal of the appendages is a sufficient reason for the removal of the uterus, because it is absolutely useless, and a large proportion of these uteri subsequently become cancerous. I was glad to see that Dr. Penrose took the ground against this extreme position. Hysterectomy is undoubtedly more dangerous than ovariotomy. If the uterus is not diseased and there is no malignant history in the family, other things being equal, I should be loathe to remove it. If the uterus were diseased and enlarged, if there were adhesions, if the healthy peritoneal covering were removed, leaving a large bleeding surface favoring the absorption of septic matter, I think that removal of the uterus would be perfectly justifiable. Most of these cases of pelvic inflammation are due to

one of two causes—gonorrhœa, or post-puerperal septic trouble. In both classes we have endometritis as the primary lesion. This may extend into the tubes without involving the deeper structures of the uterus, but as a practical matter in many long-standing cases the walls of the uterus are involved, and months or years will be required for the organ to regain its normal proportions after section. In a not inconsiderable number of these cases of complete removal of the ovaries and tubes, the patient comes back in the course of a few months with precisely the same symptoms as before operation. In a certain number of these cases I have found large uteri with thick walls and leucorrhœal discharge. In some of these patients the uterus has been removed and a complete cure effected.

The only thing that would contraindicate the operation is the mortality. Is the operation as safe as double ovariotomy? The gentlemen who have done this work have found that the mortality is no higher than in their own work in double ovariotomy for the same conditions. It is not fair to compare the results of different operators in this connection. The comparison must be made in the work of the same operator. I have done twenty or thirty hysterectomies in this class of cases without a death. The largest mortality in the past in my work has been in this same class of cases where I removed the appendages and left the uterus. This has convinced me that hysterectomy is a safer operation, because we select a class of cases where the element of time does not materially enter and the operation lessens the chances of sepsis, in addition to giving a surer opportunity of cure.

Dr. Charles P. Noble: I have been much interested in the paper, although I have not looked upon the subject from the same standpoint as has Dr. Penrose. It has seemed to me that hysterectomy, broadly speaking, was a more serious operation than we have been accustomed to do; and in order for the operation to come into popular use, its justification will be the results attained by those who have done it. As to the results obtained in the past by the methods heretofore in use: some time ago I had occasion to look over the results which I had obtained in my earlier cases. In the first seventy-five cases of double ovariotomy for pelvic inflammatory disease, instead of finding a large number of women who continued to complain of the same symptoms as before, I found only four. At least two of these were neuropathic subjects, and it was a question whether or not the symptoms had any connection with the pelvic organs at all. There were probably only two out of the seventy-five where the operation failed to relieve the symptoms. An operation which is to relieve only 2 or 3 per cent. more of the women does not fill a very large field.

I think that there is no question where the uterus is chronically diseased and much enlarged, it would be wiser to remove it, because chronic processes are slow in undergoing resolution. In acute cases, however, my experience has been that the uterus rapidly undergoes post-climacteric atrophy. If there has been some endometritis this has been relieved by careful curetting.

With reference to another class of cases. Where the uterus is retroflexed and adherent there is no doubt that often a cure is not effected unless hysterorrhaphy is added to removal of the appendages. I think that the conservative judgment of operators will be, that in such cases it is wiser to remove the uterus than to leave it stitched to the abdominal wall.

Further time and experience will be required to enable us to come to a correct conclusion concerning the good that will come from this proposed operation. Personally I think that it will not be done as frequently as its advocates at the present maintain, but that it has a limited field of usefulness.

Dr. George Erety Shoemaker: In sifting the evidence upon which to base a conclusion that heretofore not enough tissue has been removed, it is necessary to study the uncured cases.

A considerable number of these which are now going about disappointed, from one physician to another, belong to the class of neurotic or pain cases which some years ago operators considered it wise to subject to the knife, but which we have now learned should be treated by other methods, such as rest and feeding. The conclusion from these is certainly not that the uterus should also have been removed, but that no operation should have been done.

In another large division of the uncured, though they were originally suitable cases, the difficulty lies in post-operative adhesions of bowel or omentum, which by traction cause various reflexes.

Removal of the uterus, in addition to that of the tubal and ovarian masses, would not have appreciably diminished the surfaces favorable for adhesions.

We have certainly learned in the past ten years that in pelvic inflammations the dangerous and disabling conditions are outside the uterus. My own experience and observation have been that the great majority of cases have been cured, provided the operation has been undertaken for proper conditions and well done.

Dr. Shober: It has been my experience to see cases where the tubes and ovaries have been removed for inflammatory disease of tubercular or gonorrhœal origin, return with a continued purulent discharge which resisted all treatment. I agree with the reader that in cases where the uterus is infected with tubercular or gonorrhœal poison, it is wise to remove the uterus.

In those cases where there is inflammation of the ovaries and tubes which cannot be considered specific, it would be wiser to fix the uterus to the abdominal wall. I admit that where the uterus has been bound down and where the peritoneal covering of the organ has been destroyed, it may be wiser to remove the uterus. I believe that there can be a simple inflammation of the ovaries and tubes that has its origin in inflammation of the appendix. It has been clearly shown that there is an intimate relation between the blood and lymph supply of the right ovary and appendix. In such cases

it would be wiser, after removing one or both ovaries, to perform hysteropexia.

Dr. Noble: I should like Dr. Penrose to state whether the uterus is removed entirely or whether the cervix is left, and also his rules with reference to drainage in these cases.

Dr. Penrose: I have used drainage in none of these cases. Where the cervix is diseased I amputate at the vaginal junction; where it is not diseased at some convenient point below the internal os.

I agree that statistics will determine whether or not the operation is to be adopted. There necessarily will be a great difference of opinion as to the propriety of the operation in a certain class of cases. In those cases of retroflexion where there are no adhesions hysterorrhaphy may be preferable to removal, but where the fundus has been adherent more immediate and certain relief is afforded by complete removal of the organ.

SPEECH WITHOUT A LARYNX.

By HARRISON ALLEN, M.D.,
DIRECTOR OF WISTAR INSTITUTE, UNIVERSITY OF PENNSYLVANIA.

[Presented March 7, 1894.]

I HAVE the honor to report the following facts in the case of Daniel Hickey. This man talks after the removal of the larynx. The operation was performed for epithelioma by Dr. J. Solis-Cohen at the Jefferson College Hospital, April 1, 1892.[1] Dr. Cohen informs me that the parts removed included the entire larynx (excepting the free portion of the epiglottis). The first ring of the trachea was removed with the larynx, and the second was stitched to the skin of the front of the neck.[2]

The patient was mute after the operation for nearly a year, when speech returned. Inspection of the external part of the neck showed a T-shaped scar extending from the tracheal opening to near the position of the hyoid bone. The greater part of this region was occupied by a sac of irregular shape, measuring about 45 mm. in length by 26 mm. in width, whose anterior wall was the integument, and which could be easily pressed back against a resistant surface, which was doubtless that of the cervical vertebræ. The skin, which in part was occupied by scar-tissue, was perforated near the junction of the level of the old transverse and longitudinal incisions by a minute fistulous track. Bubbles of air and a little mucus

[1] See Trans. Phila. County Medical Society, 1892, vol. xiii. p. 302; also TRANSACTIONS OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA, 3d Series, 1893, vol. xv. p. 131.

[2] A brief reference to the operation will be found in the Medical News of July 23, 1892, p. 98; and a reference to the subsequent course of the case in the same journal of February 18, 1893, p. 195.

escaped from this opening. Owing to the prominence of the bellies of the sterno-cleido-mastoidei muscles the sac was in a depression whose lateral boundaries were not fixed. The sac-wall apparently had no muscular power, and was exceedingly flaccid. It will be seen that it moved freely from slight forces exerted within the mouth and pharynx. Its size varied only with the amount of air or fluid it contained. The tracheal opening was red in color, and apt to be coated by scales of inspissated mucus. The position of three rings of the trachea could be made out by the uneveness of the tracheal surface rather than by any contrasted effects of colors. The trachea moved slightly (synchronously with the pulsations of the heart) from left to right.

Examination with a laryngeal mirror, placed as in the position usual for laryngoscopy, showed the base of the tongue and the epiglottis to be normal. The epiglottis was large, broad, and but slightly depressed in the centre. It stood erect. A deep, funnel-shaped depression, answering to the interior of the sac, extended from the pharynx downward. No details could be detected within the sac other than a small scar-like structure at the posterior border of the aperture. This appeared to be most distinct at the time of a deep inspiration. I speak here of the *time* of the inspiration, and not of the assumed passage of air from the pharynx into the sac. When the nostrils were closed, the mouth open, the tongue protruded, and (as was often the case) a film of mucus was stretched across from the tongue to the velum palati, it was interesting to note that the film would persist for an indefinite period; thus proving that no air entered the sac during respiration. Forcible closure of the nostrils would often cause the film to bulge forward and break. I assume that the displacement of the air from the nose had slightly compressed that in the pharynx. The sac was seen to be easily closed by pressure from without, the two lips came together, and the space was obliterated. The fistulous track through the wall of the sac was valvular. A probe passed through it was easily discerned in the pharynx. The outer end of the fistula being covered with plaster, and

the nostrils and mouth closed, the sac could be distended so as to become convex anteriorly. I assume this was caused by elevation of the base of the tongue and velum, and consequent displacement of the air of the mouth and pharnyx downward.

The respiratory phenomena, although not subjected to precise experiment, appeared to be normal. The patient breathed from nineteen to twenty-two times per minute. The chest-expansions were free and unimpeded. A tambour being held in contact with a lever placed on the sac, and so arranged as to transmit impulses to a second tambour so disposed as to have its movements recorded on a kymographion, was found to make a downward tracing during the act of respiration, while the form of the sac did not change in shape. I conclude that these movements were secondary. The changes here noted were due to the motions of the neck itself during the act of respiration, and were probably related to the elevation of the clavicles and the upper part of the thorax. (Fig. 1.)

FIG. 1.

Respiratory curves made by motions transmitted by the neck to level placed on sac.

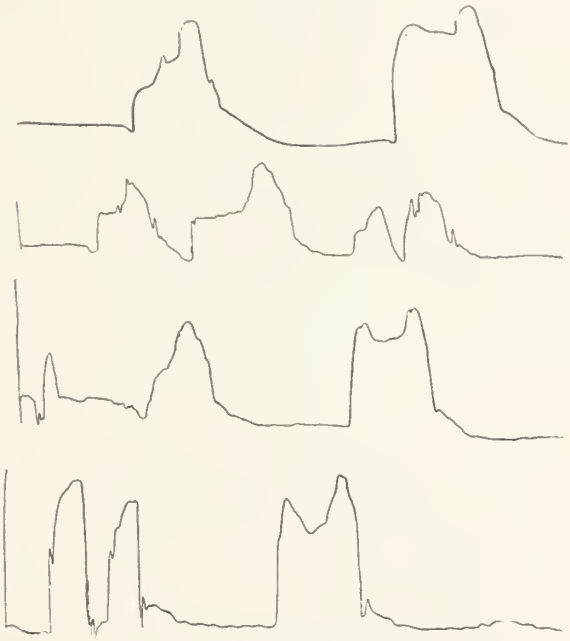
The act of swallowing was performed with comparative ease, though food was apt to pass into the sac. Liquids flowed out readily from the fistula. Pressure on the sac with the finger prevented the passage of both solids and liquids, and this pressure was regularly employed by the man in eating and drinking. The tracing made on the kymographion varied exceedingly, and appeared to be due to inconstant use of the tongue and pharyngeal muscles. Some of the tracings of deglutition are herewith given. (Fig. 2.)

The voice appeared to be true, as opposed to a whisper. It was thin, harsh, and weak, though estimated by Dr. Cohen to be heard at forty feet. Wheezy sounds of air escaping from the lung occurred coincidently with speech.

The voice was fairly well modified and was capable of slight shades of expression. The patient could sing a song, and

while the musical character of the performance was *nil*, the difference in tone between the attempt at singing and ordinary utterance was evident.

FIG. 2.



Tracings of the variants of deglutition.

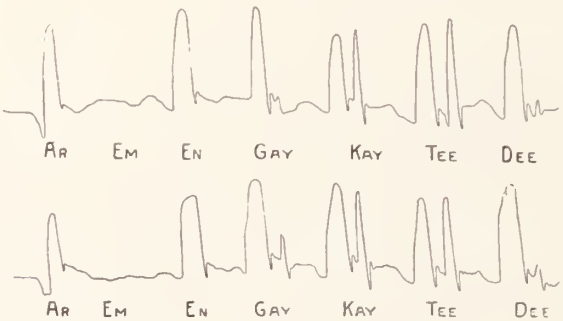
In studying the phenomena of speech the first experiments were directed to the tongue and velum. I repeated the observations of Dr. N. Kingsley to ascertain after what manner the tongue was used in articulation. I secured the assistance of Dr. W. E. Christensen, a dentist in this city, who kindly prepared a number of vulcanite plates. Coating one of these with a mixture of alcohol and prepared chalk, and placing it in the roof of the mouth, I directed the patient to pronounce the letters of the alphabet. The tongue wiped away the chalk and exposed the black color of the rubber beneath. It was easily ascertained that the contact of the tongue against the velum and palatal vault was made in a normal manner, the only dif-

ference between those thus obtained and those made in the mouth of Dr. Christensen himself being of a character that could be readily explained by individual peculiarity. The patient was rendered mute by the insertion of the plate, but by a little practice the voice returned. The slightest pressure of the plate against the velum immediately destroyed the power of utterance.[1]

The voice was next recorded on a phonograph. This was done with the object in view not only of eliminating the tracheal noises, but of catching shades of distinction of the voice that could not be detected by the unaided ear. I regret to say that these experiments were without results.

The third series of observations consisted in careful inspection of the sac during enunciation.[2] The following facts were elicited: Just before speaking the patient inspired; then at

FIG. 3.



Tracings of consonants *r*, *m*, *n*, *g*, *k*, *t*, *d*—Continental pronunciation.

the time of talking (the pronunciation of definite simple sounds was practised) the sac filled and emptied. The levers with tambours were here used, and their movements were recorded

[1] At first sight this observation would prove that the velum was an important factor in Hickey's speech. But we must recall the fact that the same effect (namely, temporary loss of speech) followed the insertion of the plate even when no impingement on the velum occurred. It is quite likely that with a little practice the presence of the plate could have been tolerated. If the free border of the velum had compensated in any degree for the loss of the vocal cords, the voice would have had a nasal quality. This was not the case.

[2] All these studies of the sac-movements were made with the opening of the fistulous track covered with adhesive plaster, so that no air entered the sac from without.

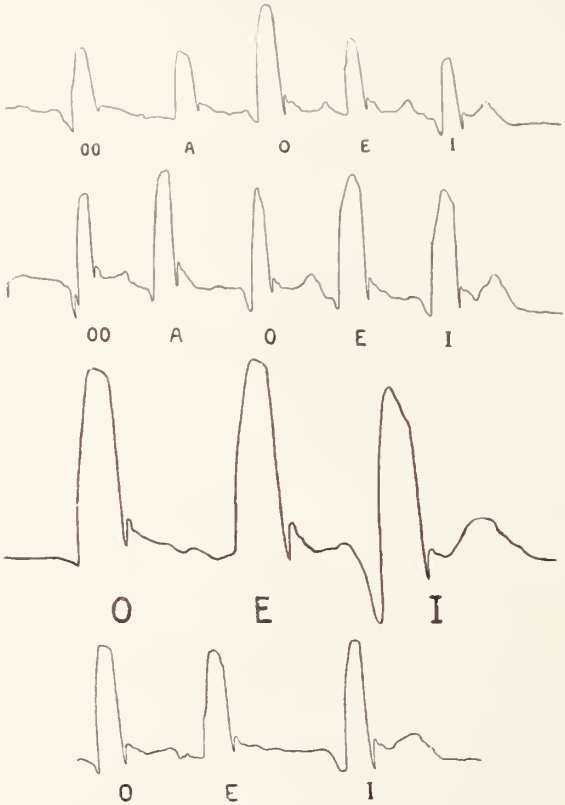
on a kymographion, as in the case of the respiratory movements. All of the speech-tracings were upward, and were thus quite unlike those of respiration. (Fig. 3.) Filling of the sac occurred in all the consonants in which *e* (*i. e.*, the sound made in the English alphabet) entered. *G* (gay) produced, as was expected, a maximum amount of movement in the sac-wall. The least constant movement occurred in *m* (em). While engaged in repeating these sounds it occurred to me that it would be convenient to have a number of phonetic characters arranged in the order of their formation from the throat toward the teeth, and I accepted the following as fairly representative of such a series: *r*, *m*, *n*, *g*, *k*, *l*, *s*, *t*, *d*. All observations were made with the patient reading aloud these letters from a chart placed in front of him, while the head was supported to eliminate all body-movements. It was soon noticed that the movements in *m* were the least constant, and the sac-movements were influenced by the act of respiration. If a full inspiration were taken, and *g* pronounced and *m* followed, scarcely any motion ensued in the sac, while if, after a full inspiration, *m* was at once pronounced the sac-walls moved almost as freely as in the pronunciation of any other sound. At the same time it was evident that while neither the air of inspiration nor that of expiration was used, the patient could speak only during expiration. I recognized the subject to be complex. The difficulties in maintaining exact uniform contact of the lever against the sac were considerable, and had not been overcome at the time the experiments were interrupted. But this much can be said: that the movements of the sac-wall were freest in the formation of the sound when the base of the tongue is known to assume the largest size (*i. e.*, humping upward), and that they were limited in range or entirely absent when the base of the tongue was known to move upward the least in the manner described; therefore, that the times of enunciation and those of movements of the sac were synchronous, and that a direct connection existed between the movements of the sac and those of the tongue. In a word, the movements of the sac-wall were due to com-

pression of air in the sac, due in its turn to displacement of air in the mouth and pharynx. (Fig. 4.)

Fig. 4.

Tracings of the same consonants illustrated in Fig. 3.
The crosses represent the times of expiration.

Fig. 5.



Variants of the tracings of the vowel sounds.
Continental pronunciation.

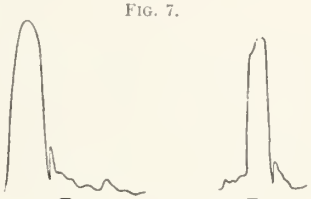
Enunciation of the vowels permits air to pass through the mouth through relatively wide apertures as compared with

enunciation of the consonants. Among the vowel-tracings I secured, as a rule, a single curve. (Fig. 5.) The variations were sufficient to lead one to suspect that oscillation was a disturbing factor; yet, when allowance is made for this error, the tracings are seen to be broadly contrasted with those of the consonants, and the semi-vowels *l* and *r* to be (as they should be) quite like the vowels themselves. (Figs. 6 and 7.)

FIG. 6.

Variants of the letter *l*.

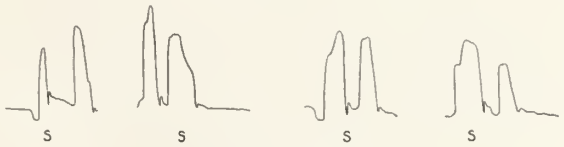
FIG. 7.



Variants of the letter *r*.

Enunciation of the consonants permits air to pass through the mouth with difficulty, if at all, and we find with them a disposition to form a double tracing, excepting in the case of *m* and *n*. The fact that *n* sometimes has a semi-vowel value probably accounts for its curve being at times like that of a vowel. Further investigation will be demanded to settle this point. I thought I had secured a uniform, constant value for this letter, but I may have been mistaken. As a rule, it may

FIG. 8.



Variants of the letter *s*.

be said that the greater the resistance to the outward flow of air the more striking become the sac-movements. As can be surmised, these were greater with surds than with sonants, and therefore were greater with *t* than with *d*, and were marked with the sibilant *s*. (Fig. 8.)

Stimulated by these observations, I was about to institute a

carefully devised series of tracings based on the tenets of phonology, when the investigation came to an end.

The relation which existed between the distention of the sac and the movement of respiration and in pronouncing *m* remains unexplained. The same may be said of the following curious observation: Occlusion of the tracheal opening, care having been taken to avoid any pressure on the sac, at once arrested speech. I tried this experiment many times. Again, occlusion of the sac likewise arrested speech. I unfortunately failed to observe if this could be overcome with practice, as was the case with the use of the vulcanite plate, or whether a whisper might have been developed when the sac was closed.

The views entertained as to the significance of articulate speech make it necessary that a current of air should vibrate against a flexible hem or border. Dr. Cohen informs me that he thought the scar-like band on the posterior aperture of the sac might serve this purpose. I see no reason why this might not at least assist, while the general constriction of the circular fibres of the inferior and middle pharyngeal constrictors might act as the cause of the speech by the air vibrating against lips of muscle-ridges which existed only at the time that enunciation occurred. The motions of the sac were also to be explained by these constrictions and (as already stated) by the tongue. But why the act of respiration is in any way related to the performance of these acts is not apparent, unless we assume that the muscle-movements are part of a complicated mechanism which demands the participation of the respiratory act as well as the pharyngeal and oral mechanisms, even in the accidental circumstance of the laryngeal apparatus being absent.

The hypothesis that at first sight is reasonable, that the sac fills when the lung fills, and that sound is produced while the sac empties itself during expiration, is in harmony with the physiology of normal speech. But I cannot see how this opinion can be entertained if it be conceded that the sac did not fill during inspiration, and that the respiratory act did not affect it any more than it did any other part of the neck. But the movements of the sac were coequal with the times of enun-

ciation, and while influenced in a measure (as in pronouncing *m*) by the act of respiration, were not dependent upon it. The idea that speech may have occurred during inspiration is suggested by the shape of the respiratory curves on the kymographion, but this was at once invalidated by the fact that the tracheal noises of expiration were always synchronous with speech, while the respiratory curve was downward instead of upward.

I venture to express the opinion that the displacement of air from the pharynx by the constriction of the lower pharynx was nearly normal, and the humping of the lingual base absolutely so. That these produced so great a change in the sac of our patient was due to the absence of a check-mechanism, the larynx itself, which thus acts in the norm as a barrier to the retrocession of the compressed air. In health the vocal bands when adducted form an almost complete barrier to the entrance of air, and in order that the bands themselves be spared from pressure, laryngeal ventricles are provided to receive and hold it. Not only is this the case, but, in the act of swallowing, these sacs may also serve a purpose of accommodating the air, which, being pushed in advance of the ingesta, would be thrown into the trachea were not a special mechanism provided. The muscle constrictors at the laryngeal orifice exist for this purpose in health. It is noted that the epiglottis is not spoken of in this connection as a factor, either in speech or as an aid to prevent the entrance of food into the larynx. It is my opinion that if it held such an attitude it would have exerted some influence in protecting the sac against the entrance of both solids and liquids in the act of deglutition.

While making the statement that the patient had voice, it is tenable that it was a voice created by the presence of an adventitious resonating chamber established in the region of the larynx, and that the utterances were of the nature of whispers reinforced by the air in this sac.

DISCUSSION.

Dr. J. Solis-Cohen: There are two or three points that I should like to comment on. A small fistula remained after cicatrization of the cutaneous wound. When the man began to speak the little fistula grew larger and larger, but the patient would not allow us to do anything to it, for he insisted that the moisture from the saliva which oozed through it facilitated his speech. When I reported the case I alluded to the change of pitch in his voice, and took it for granted that there must be some muscular substance in the new phonal reed that could be made tense to account for the differences in tone, and this, from the image in the mirror, I took to be the horizontal fibres of the inferior constrictor muscle of the pharynx on the left side of the body. The difference in the singing pitch is not more than three or four tones, and, as mentioned by one of the Fellows present on that occasion, it is largely a question of time simulating the change in pitch. His time in singing is good.

I appreciate fully the observations made by Dr. Allen with regard to the resonance in the cavity of the pharynx in reinforcing the voice. The ventricle of the larynx is a resonator of the normal voice, and in some species of howling monkeys and other animals largely reinforces the voice. So may the pharynx do in this abnormal voice.

Dr. Allen mentioned that when the tracheal opening was closed the man could not speak. I think this might largely be attributed to habit, because we are in the habit of using the auxiliary muscles of the chest in speaking. It is hard to understand how stopping the trachea should interfere with speech, as the patient does not use the air from his lungs at all.

Dr. G. G. Davis: The difference in the pitch in the human voice does not depend solely on the action of the vocal bands for its production; the uvula and soft palate likewise are important factors. Therefore cannot the slight difference of pitch which is observed in Hickey's voice be accounted for by the modifying influence exerted by the uvula and soft palate. It was only the larynx that was removed, while the palate was left undisturbed.

Dr. Harrison Allen: With reference to the observations on closure of the trachea in this man, it is, of course, possible that I may have been deceived. Hickey was tested under many conditions, with but one result. The acts of speech are under the control of the lingual pneumogastric and accessory nerves. If a single link is dropped out of its delicate mechanism speech would go on pretty much as though they were all present. I do not see why we may not accept as an hypothesis that Hickey talked during the times of escape of the air of expiration because it is natural so to do in health. He thus continued to do what was instinctive, although the necessity no longer existed.

EXCISION OF THE ENTIRE RIGHT CLAVICLE FOR TUMOR.

By JOHN ASHHURST, Jr., M.D.

[Communicated March 7, 1894.]

I HAVE here a right clavicle, the seat of an ossifying enchondroma, which I removed from a lad by excision last Saturday, March 3d. The patient was supposed to have received an injury ten months before. The growth made its appearance some months afterward, and grew quite rapidly. It was the seat of intense pain.

The specimen is rather a rare one. As far as I know there are only thirty-seven cases on record in which the whole clavicle has been removed at one operation. When done for necrosis the operation is an easy one. When done for tumor it is more difficult, the difficulty varying with the size and nature of the growth. A partial excision may be more difficult than complete excision. Some years ago I removed two-thirds of the clavicle for sarcoma, and found the operation much more difficult than the removal of the whole bone in this case. The fact that this tumor was comparatively hard and not infiltrating, made the operation easier. [The patient had no unfavorable symptoms after the removal of the bone, and was discharged, with the wound healed, on March 22d.]

VITILIGO INVOLVING THE WHOLE SURFACE OF THE BODY.

By HENRY W. STELWAGON, M.D.,
CLINICAL PROFESSOR OF DERMATOLOGY IN THE JEFFERSON MEDICAL COLLEGE.

[Exhibited March 7, 1894.]

Dr. H. W. Stelwagon exhibited a full-blooded Georgian negro with vitiligo involving the whole surface of the body.

The disease began forty or fifty years ago when he was fifteen years of age and developed slowly for fifteen or twenty years. During the past few years the progress has been more rapid, and with the exception of a few small areas on the face, the entire surface is now involved. Partial vitiligo in the negro is quite frequent, but its development to such an extent as shown in this case must be extremely rare.

CÆSAREAN SECTION AND SYMPHYSIOTOMY FOR THE RELATIVE INDICATIONS.

With Report of Cases.

By EDWARD P. DAVIS, A.M., M.D.

[Read April 4, 1894.]

The problem of successful delivery to mother and child in complicated labor derives new interest from the results of modern obstetric surgery. Obstetricians have, at the present time, better grounds than formerly for the hope that such cases may be terminated with but little mortality and comparatively little suffering. Symphysiotomy under antiseptic precautions is as yet sufficiently new to render of interest a report of its results, especially as affording a basis of judgment in determining its scope. It is the purpose of this paper to report two symphysiotomies for the relative indications, and a third case in which symphysiotomy was declined and the Cæsarean section chosen in its stead. These contrasted cases will lead to the discussion of the reasons for choosing the operative procedures mentioned.

By the phrase "symphysiotomy for the relative indications," we mean the performance of the operation in cases where it would have been possible to extract a dead child by craniotomy without opening the mother's symphysis, and where some might claim that a difficult and tedious application of the forceps might possibly have resulted in the birth of a child capable of surviving, without the operation of symphysiotomy. It is my belief, however, that it is the duty of the obstetrician,

whenever possible, to avoid difficult extraction with the forceps, not only on account of the fœtal mortality which sometimes results, but also because of the fœtal morbidity, especially in injuries to the skull and brain which not infrequently follow such delivery. To my mind, cases where there is a strong probability that the fœtus will sustain serious injury in delivery, as evidenced by failure of the fœtal head to engage after sufficient uterine contractions have persisted for a reasonable time, call for surgical interference in the interests of mother and child. The cases of symphysiotomy are briefly as follows:

Case I.—J. McN., aged twenty-five years, an Englishwoman, entered the Jefferson Maternity during last autumn. Her previous history was not obtainable at the time because of the patient's reticence and intentional misstatements; it has been learned, however, that she is married, and that five years ago she bore a small, ill-developed fœtus after a spontaneous labor lasting five days; she has also had an abortion at four months.

On admission she was found to be in the eighth month of pregnancy, the fœtus occupying the first position, the head at the brim of the pelvis and capable of being brought just within the brim by pressure; it could not, however, be made to engage. Her pelvic measurements were as follows:

	cm.
Anterior superior spines	24
Crests	25
Trochanters	27½
External conjugate	16
Internal by-measurement	7¼

The fact that the head of the fœtus could not be made to engage led me to omit the induction of labor, and to allow the patient to go to term, with the expectation of delivering her, should the head then not engage, by suitable operative interference. She engaged in housework with the other patients, and was carefully examined at intervals of ten days or two weeks, when it was found on each occasion that the head could be brought to the brim of the pelvis, but could not be made to enter the pelvic cavity. The fact that the head presented at the pelvic cavity led me to believe that but little enlargement of the pelvis would be needed to accomplish its delivery. On December 22d, labor began, the uterine contractions being regular and of considerable force; labor progressed with increasing pains, the membranes ruptured, but complete dilatation was tardy, and the pains were ineffectual in causing engagement. The os was accordingly dilated with Barnes' bags, the patient anæsthetized with ether, and an effort made to

cause the head to engage by suprapubic pressure and uterine massage. As these efforts failed, and the heart-sounds of the fœtus indicated that the child was in excellent condition, symphysiotomy was performed. It was necessary to dissect away the recti muscles to gain free access to the joint, and also to completely sever the subpubic ligament; the method employed was by an incision above the pubes, terminating three-quarters of an inch above the joint, thus leaving an unbroken cutaneous surface directly over the symphysis. The joint was severed by the Galbiati knife inserted from below, traction being made from below upward and from in front posteriorly. The pubes separated three-quarters of an inch after section; the head engaged in the left oblique diameter, the occiput behind, and was readily delivered by Tarnier's axis-traction forceps; the pelvic floor and perineum were uninjured. A strand of iodoform gauze was carried behind the symphysis to prevent the bladder and adjoining tissues from being pinched between the parts of the symphysis as the pelvis was brought together. The wound was closed in the usual manner, an open stitch being left at the lower end, which was tied when the gauze was removed twenty-four hours after operation.

The patient made a good recovery, retarded only by gastric irritability arising from her previous excessive indulgence in tea. She was kept recumbent four weeks, an antiseptic dressing being applied over the wound, and antiseptic absorbent pads over the vulva, with a single strip of adhesive plaster eight inches wide drawn tightly around the pelvis; the centre of the strip being over the trochanters of the femora. She was lifted out of bed daily, and care taken to prevent irritation of the skin and maintain cleanliness. Her child was a well-developed male, 46 cm. long, weighing 7 pounds 11 ounces; the measurements of his head were as follows:

	CM.
Occipito-frontal	11½
Occipito-mental	14
Maximum	13
Bi-parietal	11
Bi-frontal	9
Trachelo-bregmatic	9¾
Sub-occipito-bregmatic	10
Bis-acromial	11
Circumference	36

Since her convalescence the patient has been kept in the Maternity, employed in domestic service, to accurately observe the condition of her pelvis. She has been examined by Dr. R. P. Harris and by Dr. H. Augustus Wilson. She has firm and complete union at the symphysis, whether osseous or fibroid cannot be stated. She experienced, when first allowed to get up, some lameness in the right sacro-iliac joint, which has since disappeared. She is able to carry coal up and down stairs, to scrub and lift as well as other women in

the Maternity who have not been subjected to symphysiotomy and who are not in a pregnant condition. She has nursed her child, and is in good general health. The measurements of her pelvis at the present time show a slight increase, possibly as the result of symphysiotomy: thus a very careful measurement shows that in the distance between the crests of the ilia and between the trochanters there is an increase of ½ cm. Her external conjugate is 1 cm. greater than before symphysiotomy. Her internal conjugate is ¾ of a cm. greater. According to her own calculation, and from the appearance of the fœtus, she had gone several weeks over the period of normal gestation when delivered.

Case II. — M. K., a young married woman, aged twenty years, whose previous history was negative; she did not know the date of her last menstruation, and had never recognized fœtal movements. Her general development is poor; she was poorly nourished when admitted to the Jefferson Maternity and manifested an unstable condition of the nervous system. An examination of her pelvis gave the following measurements:

	CM.
Anterior superior spines	22½
Crests	25
Trochanters	27
External conjugate	19
Internal conjugate	9½

The fœtus occupied the first position, the head presenting. The period of her pregnancy was the end of the seventh and the beginning of the eighth month. An examination of her urine revealed a deficiency in excretion. The percentage of urea remained below the average, the specific gravity of the urine being less than usual, although albumin and casts were absent. She remained in fair health until the 7th and 8th of March, 1894, when she manifested symptoms of toxæmia; she complained of headache, disordered vision, great restlessness, exaggerated melancholia, and nausea. An examination of her urine for several consecutive days revealed a rapid diminution in the percentage of urea, although albumin was present in a mere trace, and casts were absent. The amount of urine diminished progressively. The percentage of urea, which should have been from $1\frac{4}{10}$ to $1\frac{8}{10}$, fell as low as $\frac{1}{10}$ of 1 per cent. Instructions were immediately given that the induction of labor should begin; this was accomplished by the introduction of bougies, uterine contractions occurring, characterized by excessive pain. The patient was given calomel in small and repeated doses, a hot bath and pack, fed upon skim milk, and made to drink freely of water, when the percentage of urea rose to $1\frac{4}{10}$. After forty-eight hours of very gradual labor by the stimulus of bougies in the uterus, the patient came into active labor on the 16th; her pains were vigorous, and caused great suffering, dilatation being tardy. Barnes' and McLean's dilators were employed, and dilatation being complete, the patient was anæsthetized with ether, the membranes were ruptured, and

an endeavor was made to bring the head to engage; this could not be done; the head presented, the occiput posteriorly and upon the right side. After a fair trial had been given to secure engagement, symphysiotomy was performed as previously described. In this case, as the disproportion between the head and the pelvis was much less than in the previous case, the subpubic ligament was but partly severed; the head at once engaged, although the parietal bone presented at the brim of the pelvis. An effort was made to apply Tarnier's forceps to the sides of the head, but they could not be readily locked. Simpson's forceps were then applied with the axis traction tapes, and the head gradually brought to the floor of the pelvis, where incomplete rotation occurred. The occiput was delivered posteriorly, the pelvic floor being uninjured. The child was asphyxiated, but was revived without great difficulty. Its head at birth measured:

	CM.
Maximum diameter	12½
Sub-occipito-bregmatic	10
Occipito-mental	12½
Occipito-frontal	11
Fronto-mental	9
Bi-parietal	9
Bi-temporal	9
Bis-acromial	11
Total length	46

Sex, male.

Weight, 5 pounds 11 ounces.

The child was placed in an incubator and fed with breast-milk pumped from the mother's breasts and dropped into the mouth with a pipette.

Eighteen hours after delivery the mother was seized with eclamptic convulsions; as she had received $\frac{8}{30}$ of a grain of strychnine hypodermatically in divided doses, and as her convulsions were not typical, there was doubt as to their precise nature. Dr. Hare saw her in consultation, and observed no evidence of strychnine poisoning. Under the use of a hot pack her convulsions ceased and did not return. The percentage of urea remained nearly normal, and has risen to normal, and her convalescence has been uninterrupted. She is able to nurse her child, which is developing normally. Her wound has healed, although she has not yet been permitted to assume the erect posture.

In contrast with the foregoing is

Case III., where the Sänger-Cæsarean section was chosen instead of symphysiotomy. Mrs. E. H., aged twenty-seven years, of excellent general health and good physique, married at eighteen; had been pregnant five times previously. Her first, third, fourth, and fifth labors were terminated by the use of forceps, each labor resulting in the birth of a dead child. All of the children were large: the first was 23 inches long and weighed 13½

pounds; the others weighed more than 12 pounds. Her second labor terminated spontaneously, a small male child being born, although her medical attendant at the time despaired of a successful termination to the labor, and had sent for assistance, when, under strong pains, the head emerged with defective mechanism of labor. The exact manner of delivery neither the patient nor her husband can describe. The child, however, was small, but is living and in good health. The patient corresponded with me during the winter, stating that she was resident in the mining regions of the State, that she had endured repeated unsuccessful labor, and was desirous of obtaining at the termination of her present pregnancy a living child. She gladly came to the city and presented herself at my office for examination. In general appearance, physique, and width of her hips she seemed an exceptionally robust woman. It was evident that her uterus contained an unusually large child from the size of the abdominal tumor. An examination of her pelvis gave the following measurements:

	CM.
Anterior superior spines	28
Crests	30
Trochanters	29½
External conjugate	19½

Vaginal examination showed the rami of the pubis approximating each other much more closely than normal. Palpation of the inner surface of the pelvis revealed a funnel-shaped pelvis, the cavity narrowing very appreciably toward the outlet. In comparison with the breadth of the patient's shoulders and the width of the iliac bones, it was seen that she had a flattened and converging pelvis. Fœtal heart-sounds were heard upon the right side of the abdomen, four inches above the umbilicus, indistinctly over a wide area. The os uteri was patulous, but no presenting part could be detected. The symphysis pubis was two and one-half inches in length.

The patient was convalescent from grippe and had an obstinate cough. She was told that an operation would be necessary for her safe delivery, to which she assented. She was instructed to go immediately to the Polyclinic, where it was intended to prepare her for delivery by surgical means; she neglected, however, to report at the hospital for twenty-four hours, when she came in at midnight on January 14th, in labor. In the early morning of January 15th her pains became frequent and severe; the membranes ruptured and a breech presentation could be detected; the child remained high in the mother's abdomen, the breech failing to engage. Dr. Harris met me in consultation over the patient about six hours after the beginning of labor. His first advice was to perform symphysiotomy, but when palpation and auscultation confirmed the diagnosis of breech presentation, and the large size of the child was apparent, together with the fact that version could not be performed on account of the loss of amniotic liquid, Dr. Harris and my colleague at the Polyclinic, Dr. Baer, agreed with me in choosing Cæsarean section.

On account of the patient's condition of bronchial irritation chloroform was administered by my assistant, Dr. Wm. H. Wells. In listening for the heart-sounds, it was found that the placenta was attached to the anterior wall of the uterus, and would probably be encountered in the incision. The abdomen being opened, the uterus presented, large in size, and rotated from right to left upon its axis. While Dr. Baer skilfully controlled hemorrhage by taking the broad ligaments between his fingers, I incised the uterus, finding the placenta directly beneath the incision; the placenta was immediately severed, stripped from the wall of the uterus, and the child, placenta, and membranes delivered in a few seconds. The child was delivered by the feet, it was asphyxiated, but speedily revived. The uterus was turned out of the abdomen and closed by four buried sutures of heavy silk, six stitches through muscle and peritoneum, and seven fine silk stitches in the peritoneum only. There was but little hemorrhage, although bleeding from one of the sinuses required two additional stitches. The size of the uterus required an abdominal incision extending above the umbilicus, which was closed by continuous sutures through the aponeurosis, and fourteen interrupted stitches. The patient showed little shock, the uterus contracted well, and there was no secondary hemorrhage or relaxation. The child was a female, weighing $11\frac{3}{4}$ pounds, and 52 cm. long. The measurements of its head were as follows:

	CM.
Occipito-frontal	13
Sub-occipito-bregmatic	$11\frac{1}{4}$
Bi-parietal	$10\frac{1}{4}$
Bi-temporal	$9\frac{1}{4}$
Occipito-mental	14
Maximum	15
Bis-acromial	$12\frac{1}{2}$

The mother's convalescence was jeopardized by two complications: her cough continued obstinate for several days, and catarrhal pneumonia seemed threatened for a short time. This condition gradually subsided. Her failure to report promptly at the hospital had given insufficient time to disinfect the surface of the abdomen, and a single stitch-hole abscess required attention for a few days. With these exceptions, her convalescence was uneventful; she nurses her child, and is living in good health in the city. Because of the length of the abdominal incision the stitches were retained as long as possible in her case, as it was feared that violent cough might reopen the incision.

This case is of interest in connection with the preceding, because symphysiotomy was declined for two reasons: first, the funnel shape of the pelvis promised less in gain of space after symphysiotomy than in pelves shaped as in the preceding cases; second, Dr. Harris informed me that breech presentation is less favorable for delivery after symphysiotomy than head presentation, and as the membranes had ruptured, it was impossible to turn the child before operating. If I remember correctly, Dr. Harris was positive, on see-

ing the child, that it could not have been safely delivered in breech presentation after symphysiotomy. Five weeks after delivery, examination of the patient revealed the following condition: the cervix uteri pointed downward and slightly backward; the uterus adherent to the abdominal wall, reaching a hand's breadth above the pubes. Involution of the uterus had proceeded well; a slight amount of mucous secretion was present from the cervix.

In conclusion, the report of these cases is offered as an illustration of what the writer believes to be a fair deduction from the results of modern obstetric surgery, namely, that in cases where the fœtus is disproportionate in size to the mother's pelvis, and where after spontaneous efforts have failed to secure engagement of the presenting part in a reasonable time, and these efforts have been supplemented by thorough examination under anæsthesia, and a fair trial to secure engagement by manipulation and suprapubic pressure, that if the fœtus be living and in good condition, it is the duty of the obstetrician to refrain from application of the forceps only, and to deliver the patient by some form of abdominal section, either symphysiotomy or the Cæsarean operation.

DISCUSSION.

Dr. Robert P. Harris: With regard to the case of Cæsarean section. I presume I have less fear of the Cæsarean operation than almost any man in America. The cases that I have seen, where I expected the patients to recover, all did so. I know that I have witnessed more recoveries after Cæsarean section than any man in America, amounting to eleven out of thirteen.

In New York they are partial to version after symphysiotomy. I feel satisfied that if a child like the one in question, with a large round head, was delivered by the feet after this operation, it would be lost. As it was, after the Cæsarean section, the child was deeply asphyxiated, requiring some little care to get it to respire. I believe that many children are injured by forceps, many more than we have any idea of. These are cases where the pelvis is so small that a great deal of forceps pressure is required to bring the child into the world. The question is, whether these fœtuses are not safer under symphysiotomy. We often see curious elongated heads in grown people which may have been produced by forceps or prolonged labor,

moulding the cranium into such a form that it never resumed its normal shape. Because a fœtus lives under the forceps pressure is no proof that it has escaped injury.

The last twenty-five symphysiotomies in this country have been followed by death in only two instances. The two deaths were pretty much foregone conclusions. One woman had been in labor seventy-two hours. In the other case, both the mother and the child were lost. The child was dead at the time, and the mother lived only twelve hours. Symphysiotomy *per se* should have very little mortality. We have had forty-five operations in the United States with six deaths, and the death-rate is diminishing under experience. In England they condemn the operation and think that Cæsarean section is the less fatal. They have had two symphysiotomies in England and one in Ireland in one hundred and twelve years, and base their opinion upon their own results.

Dr. E. P. Davis: The view taken that where the fœtal head will not engage in spontaneous labor, or cannot be made to engage by manipulation when the patient is anæsthetized, operation is necessary, is not the result of theory, but a radical change in my own belief. I was taught that these were cases for the prolonged use of the forceps. I recall such cases where the child was lost, and where I have no doubt that the child would have been saved by a resort to symphysiotomy.

FURTHER REMARKS ON THE OCCURRENCE OF A FORM OF NON-ALBUMINOUS NEPHRITIS OTHER THAN TYPICAL FIBROID KIDNEY.

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[Read by title, April 4, 1894.]

Recently,[1] under the title "The Occurrence of a Form of Chronic Bright's Disease, other than Typical Fibroid Kidney, without Albuminuria," I directed attention to a hitherto unrecognized and apparently not uncommon class of cases of chronic nephritis, representing a type clinically distinct from that viewed as interstitial nephritis. Briefly it may be said to be one the clinical symptoms of which place it quite without the boundary of the well-known varieties of so-called Bright's disease, such as chronic desquamative nephritis, or the fibroid or granular kidney. I dwelt upon the wide difference clinically between the group of cases I described and that with which these are most likely to be confused—interstitial nephritis, the red, granular, or fibroid kidney. I pointed out that it has long been known that cases of fibroid kidney could occur, especially in the early stages of the malady, without the presence of albumin in the urine, or with albumin existing, if constant, only in traces, or to be found in certain portions of the twenty-four-hour urine. The occurrence of typical granular kidney without albuminuria, I said, had been first remarked by Wilks, of Guy's Hospital, nearly a half-century

[1] The American Journal of the Medical Sciences, December, 1893.

ago, but it was Mahomed some three decades later who especially investigated cases of this phase of the disease, describing a considerable number of them in the *Guy's Hospital Reports* for 1879 and 1880–81. I stated that it was doubtful if many of Mahomed's cases, such as I cited, were actually cases of renal disease, though they could be properly included in the category of chronic Bright's disease as Mahomed employed the term. The symptoms of many of these were in the main referable to other organs, and in a minority of those in which a necropsy was obtained the kidneys were stated to be normal.[1]

I also observed that

"Apart from the doubtful cases of renal disease among those reported by Mahomed, in which albuminuria was absent, there remains a good number in which, with the urine free from albumin, undoubted granular kidney existed. Be that as it may, Mahomed's cases were all in middle or advanced life. There were present decided cardio-vascular changes, such as hypertrophied heart and thickened vessels. These cases were reported especially to show that in the stage of Bright's disease in which epithelial alterations in the tubules were absent—or, if occurring, were but transitory in character—albuminuria was absent, the urine remaining practically normal, and symptoms referable to the kidney, *i. e.*, those of renal inadequacy, were uncommon. Death in these cases, as Mahomed remarks is usual in granular kidney, resulted from failure of other organs, notably the heart."

The class of cases described by Mahomed and subsequently referred to by Millard,[2] and especially by Purdy,[3] related only to the typical arterio-capillary fibroid kidney so ably portrayed by Sir William Gull:[4] an affection occuring in the

[1] I stated that Mahomed adopted the term chronic Bright's disease as a convenient generic name for a condition, which Gull and Sutton had previously described, of generalized arterio-capillary fibroid change. Contrary to the view of Johnson, an extension of that of Bright, and following in the lead of the above-mentioned investigators, Mahomed viewed the condition underlying the various stages of granular or fibroid kidney as a systemic disorder, in which the cardio-vascular alterations were not dependent upon, but either antedated the development of the kidney fibrosis, or more rarely existed without renal change. It was not necessary for him that the kidney be the seat of degeneration to constitute Bright's disease. Fibroid kidney was but an unnecessary, if more or less to be expected, incident in the general morbid state. Renal fibrosis did not always appear, though general arterial change occurred.

[2] Bright's Disease. William Wood & Co.

[3] "The Pre-Albuminuric Stage of Chronic Bright's Disease," Chicago Medical Journal and Examiner, May, 1885.

[4] American Journal of the Medical Sciences, 1886, vol. xci. p. 407.

degenerative period of life, and part and parcel of a diffused cardio vascular decay. In this class it is well recognized that with an accompanying polyuria, or with urine, less commonly perhaps, unaltered as regards amount, the excretion of urinary solids, especially the nitrogenous, is often not far from normal, as in these the histologic renal changes may be but slight in character, the secretory structure not being sufficiently implicated to show marked deviation from healthy function. When such deviation, however, becomes apparent, it is likely to be coincident with the appearance of decided amounts of albumin and of casts in the urine, with dropsy and marked uræmic symptoms.

Until the appearance of my paper no mention had been made by systematic writers of the likelihood of the occurrence of a non-albuminuric form of chronic kidney disease[1] other than that regarded as a typical interstital nephritis, with characteristic symptoms, although a case here and there has been occasionally reported, notably such as one recently by Reed,[2] to which my attention has just been directed, in which the few symptoms detailed suggest that it is closely akin to, if not actually identical with, the class I have described. Its withdrawal, however, from one of the time-honored categories is not thought of. As Purdy, following out the lines drawn by Mahomed, refers to such cases under the title of the pre-albuminuric stage of chronic Bright's disease, so Dr. Reed discusses the case he relates under the caption "diagnosis and treatment of the *early stages* of chronic Bright's disease." What I especially desire to emphasize is that there certainly exists a class of cases probably of common occurrence, with symptoms which place them in a group entirely distinct from what is known as chronic interstitial nephritis. In these the most aggravated symptoms of renal inadequacy may be present,

[1] Apart entirely from the disadvantages of a cognominal nomenclature, I have thought it better to discard the term *Bright's disease*, especially in referring to non-albuminuric forms; as here employed it is essentially misleading—Bright's researches referring solely to affections of the kidney having as their chief mode of recognition the invariable presence of albumin in the urine.

[2] Medical Record, New York, April 1, 1893.

such as are common only in an advanced stage of kidney disease, and yet albumin remains totally absent from the urine.

That these cases have a pathology distinct from the so-called arterio-capillary fibroid kidney (the red granular or contracted kidney, or interstitial or catarrhal nephritis) I have no doubt; but the most exact post-mortem investigation into the character and distribution of the histologic change is necessary in cases which, dying, still preserve the usual symptom-grouping in order to determine the actual differences. Probably a lack of comprehension of certain facts so lucidly expressed by the late Sir William Gull, who, in considering the pathology of fibroid kidney, so ably argued against the unity of renal disease, has done much to prevent a faithful and exact study, and a proper differentiation of the various forms of chronic nephritis.

Gull adverted to the erroneous inference so often drawn by the illogical, that, because *post-mortem* in the several forms of renal disease histologic changes are encountered common to all forms, it should follow that these are of a kind, with an identical pathology. He says it is "as though one should assert that all scars of the skin, seeing that they have largely common histological characters, have also one pathology."

It is a well-recognized fact that whatever the form of chronic nephritis, whether it have distinctly arisen in tubule, glomerulus, or in interstitial renal structure, eventually, in the later stages of the malady, the histologic changes are liable to have become so diffuse with coincident symptomatic change in the type of the ailment, that a necropsy totally fails to throw light on the exact character or anatomic seat of the original morbid condition, however limited its nature may have been for a long time.[1]

[1] In reference to this point, Sir William Gull argued that, although within limits, the morbid anatomy of the kidney is distinctive of the pathologic condition from which it originates, yet "It would seem not to be sufficiently considered that, in the nature of the case, the morbid forms of histological expression are limited, whatever may be their pathology, and hence these lines of morbid tissue-change will have a tendency to approach each other as they proceed. For example, interstitial nephritis, and its results in fibroid tissue and contraction, may occur in kidneys in which the morbific agencies may entirely differ among themselves. In catarrhal nephritis there are various grades of interstitial

In cases of the sort that I described, therefore, the exact pathologic footing must remain a matter of conjecture until a series of necropsies are obtained with death occurring before marked change in type is manifest, before generalized fibrosis has become evident, or albumin has appeared in the urine. This may be possible only after a period of years, but will be the earlier the greater the number of these cases that are recognized, collected, and followed. Light may only be thrown on the pathologic position of such cases through death from an intercurrent malady.

It certainly seems logical that a group furnishing more or less prominently symptoms so distinctive and separable from those of the well-recognized forms of chronic nephritis has a distinct pathology. With an oliguria as persistent and as marked in certain cases as in the most outspoken type of chronic parenchymatous nephritis, and with a urine as concentrated, yet with absence of cardiac debility and dropsy to indicate its source, there occur as grave continuous symptoms of renal inadequacy—those, indeed, of pronounced uræmia. These are associated, too, as in chronic parenchymatous nephritis, with as marked diminution in urinary solids, notably diminution in urea and mineral ingredients, indicating undoubted secretory involvement; yet totally unlike what is distinctive of parenchymatous nephritis, albumin is so persistently absent from the urine, even with the existence of the aggravated symptoms of renal incompetency, as to clearly indicate that its presence need form no part of the assemblage of symptoms or have any relation to the morbid condition.

The following table graphically presents the chief points of

nephritis, which may produce granulations of the organs, though such interstitial changes may have quite a different meaning from that which occurs in the fibroid kidney . . . And the same might be said of the nephritis of scarlatina, of pregnancy, and of other forms. In fact . . . in all cases of renal lesions there will be an approximation more or less to histological changes common to the whole. . . . For, however the lesion may have begun and from whatever cause it may have sprung, its results are destructive; and in the process of destruction they must approximate toward each other, and, therefore, contraction, granulation, and atrophy may occur in any nephritis, so that the exclusive morbid anatomist will readily find on the post-mortem table a strong confirmation that there is but one nephritis, one Bright's disease."

differentiation characterizing the well-known chief varieties of chronic nephritis, and the few cases of the non-albuminuric form studied:

Chronic parenchymatous nephritis.	Chronic interstitial nephritis.	A form of non-albuminuric chronic nephritis closely resembling neither.
Urine always albuminous. In amount it is scanty, except during secondary atrophy; then it may be abundant. It is light-colored, depositing urates readily. Specific gravity normal or higher than normal, though low for the total amount passed.	Urine not constantly albuminous Usually, however, albuminous in degree recognizable by HNO_3 (Heller's method). Urine profuse in amount (unless indications of cardiac failure present, with accompanying signs, dropsy etc.), light-colored, low gravity. Sediment usually slight; often none visible.	If urine is ever albuminous it is probably not as an incident of the malady, as the most decided symptoms of renal inadequacy may be present, even producing chronic uræmia, with albumin persistently absent from the urine. Urine subnormal in amount; usually quite scanty and high-colored, and this totally without evidences of cardiac weakness, and with persistent high blood-pressure and absent dropsy, indicating that the source of the oliguria is probably not of vascular origin. The specific gravity is normal or higher than normal, though relatively low for the amount of urine voided.
Casts numerous and in great variety; epithelial, granular, waxy, and hyaline. Blood-corpuscles and connective-tissue shreds not infrequent. Microscopically, urates and phosphates predominate, oxalates less common.	Casts not frequent. When detected commonly hyaline, and less often granular. Renal epithelium and blood-cells generally scanty or absent. Oxalates common.	Casts common, although, as a rule, not very numerous; principally hyaline, but finally granular present in all in less number. Epithelial casts rare; seen in very small number in two of the cases. Waxy casts noted in one. Cylindroids present in all; in several very numerous — both mucous and hyaline. Renal epithelium in all. Oxalates very common. Urates often in relative excess. Occasionally blood-cells and pus-cells.
Urea usually much diminished in amount. Uric acid practically normal.	Total solids often normal. Urea may be normal. Usually somewhat diminished. Uric acid usually diminished.	Total solids—urea and mineral salts—always diminished in amount. Uric acid normal or diminished.
Cardiac hypertrophy not invariable, although blood-pressure tends to be habitually raised. Atheroma of arteries not common early in the affection.	Cardiac hypertrophy usual. Blood-pressure habitually raised. Fibrosis of arteries almost invariable, usually to a pronounced degree.	Cardiac hypertrophy not detectable in any of the cases, although in all the first sound is more or less forcible, and in none is it weak. In all the aortic second sound is also accentuated at the apex. Blood-pressure is habitually raised in all save one. This elevation is not detectable in two while on a strictly non-nitrogenous diet; arterial fibrosis not evident in any.
Uræmic symptoms common, although often less frequent than in interstitial nephritis. No loin-pain.	Uræmic symptoms common. No loin-pain.	Uræmic symptoms common. Loin-tire, or pronounced aching, usual.
Dropsy usual and obstinate.	No dropsy, and even œdema often not detectable until disease advanced.	No dropsy; œdema not common.
Occurs most frequently before age of forty. Patient pale, waxy looking.	Occurs most frequently after forty, at degenerative period of life. Patient for a long time shows only signs of failure of nutrition. Complexion grayish.	Three of the seven cases under thirty; others at or approach middle life. None show signs of a degenerative process. Facies pale, but not marked anæmia.

The six cases previously reported agree quite closely as regards the chief symptoms and the urinary condition. In all there are more or less marked symptoms of impairment of the general health, suggesting an underlying general ailment. There is imperfect gastric digestion, as shown in several, by examination of the gastric secretions, to be of the nature of subacidity and delayed propulsion. There are headache, vertigo, loin-pain, oliguria, with diminution in the most important of the urinary constituents, and the presence of casts. In all of the cases diminution in the amount of urine is a prominent symptom. During the period in which continuous observations were made on the urine of one of the series,[1] however, the urine was nearly normal in amount, averaging a daily quantity of 1085 c.c. The season was cold and water was drunk much in excess of thirst to stimulate diuresis. Subsequently the amount of urine much diminished, and now but 500 c.c. are commonly passed. There is marked increase in uræmic phenomena. The patient became so debilitated that he was compelled to abandon his occupation, and is wintering in Algiers, without, however, deriving benefit from the change of climate. Case II., a brother of the preceding, also has steadily declined in health. The amount of urine voided is habitually low. There are now present, beside more or less constant headache, aching in the loins, prostration, spells of vertigo, and not infrequently, at night, attacks of slight convulsive seizures of unquestionably uræmic nature. Without the appearance of albumin in the urine, these symptoms were common on leaving this city for a warmer climate three months ago. Cases V. and VI., which have also been under observation, show no change from the condition noted in the previous paper. Cases III. and IV. I have, unfortunately, been unable to follow, despite every effort. Case III. was an office patient; the other was seen in consultation; she changed her residence, and has not since been located.

The following case, not before reported, which I have had

[1] Case I. W. B.: see p. 662, American Journal of the Medical Sciences, December, 1893.

under observation for some time, corresponds clinically so closely, respecting the most prominent of the symptoms, with the histories of the others, that it is worthy of study as another example of how marked the symptoms of chronic nephritis may be without the presence of albumin in the urine. Its recital will serve to emphasize the fact of the occurrence of such cases, which are doubtless very often overlooked in the consulting-room, in consequence of too blind faith being placed upon negative results reached from an examination of single unmixed specimens of urine for albumin and by the use of the urinometer:[1]

Mrs. F. S., aged fifty years, weight 110 pounds, by occupation a hospital nurse, a widow, has borne two children, one thirty-two, the other now thirty years of age; she has had no miscarriages, and has led a correct life. There is no history of any disease of childhood, save measles. There is no history of diphtheria, typhoid fever, malaria, gout, syphilis, or lead-poisoning. There is on the maternal and paternal side a history of tuberculosis pulmonum, but she has never herself shown a tuberculous tendency.

She never at any time had a serious illness until about fifteen years ago. Then she was confined to bed for some eleven months by an ailment or complication of ailments the nature of which, from the best description the patient can now give, is not clear. The most lucid description she can give of her prior condition and that of the illness mentioned, is that she had not been well for a considerable time preceding the sudden development of the latter. She had felt weary, mentally dull, and incapable of exertion, and had vertigo; all somewhat after her present symptoms, though then felt less markedly. There had been anorexia, but on the day of the onset of the illness she had excessive appetite. The same night vomiting and purging appeared. She then became very sick, too much so to now recall the exact symptoms which subsequently developed. Her physician, since deceased, diagnosticated the condition as one of "abscesses in the liver, inflammation of the kidneys, and dropsy in the left chest." The left side of the chest was at first the seat of severe pain and subsequently of enlargement. The feet and abdomen were also somewhat swollen. She recalls nothing being said as to the condition of the urine at this time. Subsequent to this attack, which, whatever its nature, was finally recovered from, she remained in quite fair health until two or three years ago, when there appeared noticeable, yet then not so aggravated, symptoms of renal disorder such as are now manifest. She can recall at no time in her life of voiding more than a moderate

[1] That is, not examining as to the daily amount, and judging, with freedom from albumin, and with normal color and specific gravity, that this practically excludes a renal ailment.

amount of urine, but believes that until the past few (perhaps three or four) years the quantity habitually passed was about normal. At all events it was never in excess of normal. Within three or four years she has been surprised at the small quantity habitually voided This has rarely exceeded a pint daily unless a diuretic was taken, and lately has been persistently less. During the winter of 1892 and 1893, and the spring of the last-mentioned year, the amount fell below a pint, and at one time, by measure, for some weeks averaged only about ten to twelve ounces. A sample was then examined by a physician whom she consulted because of her symptoms, and the oliguria. It was reported to her that the examination showed the urine normal. At this time the abandonment of work and a prolonged rest of several months in the country, with tonics, improved her condition. The urine then rose to about a pint daily, and thus continued until the autumn of 1893. She has noticed for a long time a tendency to perspire quite freely when the quantity of urine has diminished, so that probably the action of the skin in her case is very complemental, and prevents the occurrence of pronounced uræmic symptoms which otherwise might arise.

The face and ankles have had a tendency to puff slightly lately, but there has been no more than a very moderate œdema manifest. She has had, with persistent anorexia, frequent spells of nausea, and sometimes vomiting, even while the stomach is free from food. Vertigo is common, especially while on the street. Headache has been for a long time quite persistent. Though she has had symptoms of impaired digestion, the bowels have been quite regular until the past year. In this time attacks of slight diarrhœa have been common, occurring at intervals of a few months, coincidently with attacks of severe abdominal cramps and the passage of more or less large quantities of mucous casts from the bowel. When she first consulted me it was especially for relief from this condition. So little was said regarding her general ill-health during the first few times that I saw her, that it was not until the bowel-trouble was relieved that I began to suspect a latent renal affection, apart from movable kidney, which I had previously looked for and excluded.

I examined several unmixed, and subsequently mixed, specimens of her urine during the time she was under treatment for the attacks of mucous enteritis. These were always free from albumin, as tested by picric acid, and of proper color and specific gravity for a normal twenty-four-hour amount, which quantity then I did not note. During the following month (November last) the general symptoms of ill-health detailed, such as headache, vertigo, nausea, somnolence, apart from the bowel-condition, which for the time was removed, became so decided and seemed so suggestive that I began examination of the twenty-four-hour urine both chemically and microscopically. As before, I found no albumin. The urea was low and there were cylindroids and hyaline casts. Several twenty-four-hour specimens were so examined at odd times, with the same result, before the con-

secutive daily examination to be detailed was undertaken. As regards general symptoms, during these visits, I noted as follows: Cardiac examination, repeated on several occasions, showed no evidence of enlargement; the apex was normally placed; the impulse though forcible was not heaving; there were no murmurs; the sounds were clear—the second was especially accentuated, and markedly so at the apex. Signs of arterial sclerosis are absent. Though the patient is not fleshy, the temporals are not discernible, save to palpation, and then give no evidence of thickening; nor do the radials, the feel of which, however, is that of raised tension; for though some pressure with the proximal finger is required to obliterate the blood stream, when a greater amount is applied it is impossible to clearly palpate the artery by the distal finger. Appended are her sphygmograms, with which many others since taken at intervals of from a week to ten days correspond. The tracing is typically that of high arterial tension. The second shows anacrotism, in her case very often produced on increasing the amount of pressure on the pulse. This anacrotic condition here, with total absence of evidence of aortic stenosis or other cardiac disease, indicates resistance in the arterioles, so characteristic of chronic nephritis.

TRACING NO. 1.

Sitting. Pressure, three ounces.

TRACING NO. 2.

Anacrotic tracing. Sitting. Pressure, three and a half ounces.

Headache is constant. Vertigo is common when in the erect posture, and at times it is so decided as to render her gait unsteady. There is persistent slight nausea. At times vomiting occurs and is independent of taking food. She is perpetually drowsy, though she sleeps very badly and is disturbed by night-terrors. Recently dull aching in the loins has been common—a symptom constantly present in the other cases.

Dr. C. A. Oliver kindly examined her eyes, rendering me a very complete report, of which the following, having sole reference to the eye ground, is a summary:

"A well-pronounced perivasculitis in each eye, more marked in the right, which is associated with a slight concentric contraction of the visual fields, these being the only ocular changes of any pathological significance; the first symptom indicating probable general vascular disturbance of a similar type."

On being unable to detect albumin in any of the single or mixed twenty-

four-hour specimens of urine, even by delicate tests, I later began a systematic examination of daily consecutive twenty-four hour specimens over a period of thirty-two days to ascertain whether albumin might not exist on some occasions. At this time as well, the total consecutive daily amount of urine passed, the urea, uric acid, and chlorids were estimated as noted here. During this period of somewhat over a month the patient was upon ordinary diet, eating animal food once or twice daily. The precaution was always observed to void urine prior to stool. I am absolutely certain that the figures as cited represent the actual amount passed. No laxative, save a small nightly dose of cascara, was taken during this period, and no other drugs were then used.

Average daily amount of urine in 32 consecutive days, 356 c.c. or 12 fl.oz.

Average daily amount of urea in 32 consecutive days, 10.52 grammes or 162 grains.

Average daily amount of uric acid in 14 consecutive days, 0.3660 grammes or 5.6364 grains.

Average daily amount of chlorine in 14 days, 1.82 grammes or 28 grains, or calculated as NaCl, 3 grammes (46.2 grains).

Average daily specific gravity for 32 consecutive days, 1025¾.

Approximate average daily amount of the total urinary solids during the period of 32 days, 20.65 grammes or 209 grains.

The color varied from red, when the amount was below 300 c.c., to yellowish-red, reddish-yellow or yellow, when the amount was above this figure.

The urinary solids[1] in this case are very low, representing for the body-weight and ordinary diet not more than what the excretion of urea should be, while the urea-excretion is about one-half the normal. The only mineral ingredient of the urine estimated in this case was the chlorides. These were taken, as representing that found pretty constantly diminished in chronic nephritis, and also as the habitual diminution of the urinary chlorine in face of a coincident diminution in secretion of HCl in the stomach (as was ascertained here) shows unquestionably incompetent kidneys, sodium chloride remaining in excess in the blood, if ingested with food, as in this case, in ordinary quantity. Here the chlorine excretion, like that of urea, is markedly subnormal; the figure, 1.82 grammes (= 3 grammes NaCl), is less than half the ordinary output. Uric-acid excretion is apparently diminished also, though not to more than a slight degree.

The influence of various remedies in common use for their diuretic effect in cases of inactive kidneys, such as the vegetable acids salts of potassium, the solution of iron and ammonium acetate, and, later, cantharides, was suc-

[1] The total solids here approximately estimated are calculated by the very simple method of multiplying the last two figures of the specific gravity by the number of ounces of urine passed; this equals the amount in grains. If an accurate urinometer is used and corrections for temperature noted (as should always be done for whatever purpose observations on the specific gravity are made) and urea and chlorine calculated coincidently, this method furnishes results that are of the greatest clinical accuracy.

cessively tried. The alkaline diuretics and Basham's mixture were without effect. The tincture of cantharides in doses of from 3 to 12 drops, three times daily, did not influence the amount passed, which at the time lay between 240 c.c. (8 fluidounces) and 330 c.c. (11 fluidounces) until taken for a week; then an increase occurred to 480 c.c. (16 fluidounces), and thence gradually to upward of 700 c.c.—this last for one day only. This augmentation was not subsequently maintained, although the remedy was continued in varying doses. The amount gradually fell, despite the cantharides, to the old figure. Regularity of the bowels was maintained by cascara. An occasional free calomel purge was given. No marked effect was apparent in the symptoms from these procedures, save that vertigo and headache were temporarily diminished by free purgation.

From the now well-known effect of thyroid extract on the urine in cases of myxœdema, increasing the urinary water and nitrogen,[1] it seemed worth a trial in this and analogous cases with incompetent kidneys, the secretory function of which was so profoundly affected. It is not beyond theoretic probability at least that such a remedy as this, exerting a specific effect upon the secretory epithelium, might also, if sufficiently long continued in disease of the viscus, which normally it can so influence, exert more than a transitory effect for good. The patient has now taken the thyroid extract steadily for a month. It will be continued for some time should no untoward effect arise. At first the extract was administered in doses of 5 grains three times daily for a period of four days only, to note its effect upon the urine both before, during, and after using Subsequently it was re-begun in doses of from 3 to 5 grains three times daily, and is so continued.

Daily amount of urine for five consecutive days before thyroid extract, 396 c.c.

Daily amount of urine while taking thyroid extract for four days, 574 c.c.

Average daily urea-excretion before, 11.19 grammes.

Average daily urea-excretion during, 12.91 grammes.

As already indicated, a sharp diminution in amount succeeded the rise under thyroid extract, on temporarily discontinuing the latter. Subsequently the urine again increased in amount on resuming the remedy. It has since maintained itself at a fair average, running between 450 c c. and 900 c.c. There is as yet no more than slight symptomatic improvement.

On several occasions prior to the period in which the continuous daily estimations of urine were made, while no drugs were taken, throughout this time and subsequently, until the patient began thyroid extract, under which the urine became too turbid for careful testing, extended examinations for albumin of the twenty four-hour urine were made. Daily consecutive examinations were carried out, always on an identical plan, from November 29th to December 31st, save on three days in which no tests were made for albumin.

[1] See a paper by Ord and White, British Medical Journal, July 29, 1893.

It was not unusual for the urine, because of its concentration, to be turbid from urates with a room-temperature of 55° to 60° F. This turbidity occurred with about three-fourths of the specimens. The urine always cleared perfectly by heat, and no trace of cloud occurred on prolonged boiling—a process that a small portion of the urine was put through on each occasion that the turbid urine was tested.[1] A considerable number of microscopic examinations were made, chiefly of the specimens in which no great turbidity occurred from precipitation of amorphous urates after standing, but, also, in some of the latter when this condition persisted for several days. Then nearly the total bulk of urine, after the application of a gentle heat to assist in the solution of the urates, was diluted from the few ounces representing nearly the twenty-four-hour quantity to that equalling a more normal amount, and a little sodium carbonate added to maintain the urates in solution. For microscopic examination the urine was always allowed to stand in a tall glass from twelve to twenty-four hours, and the sediment thus obtained centrifugalized. On all occasions cylindroids were found and usually in large amount. On several instances the number was surprising. They were, as a rule, mucous, but not infrequently perfectly hyaline. Typical hyaline casts, both medium-sized, broad, and narrow, were often found, but were incomparably less in number than the cylindroids. From time to time a few undoubted finely granular casts were also encountered. The sediment, from the concentration of its diluent, was always highly rich in cellular elements from the extra-renal passages. There were detected in the slides of nearly all specimens examined a few cells, the appearance of which suggested origin from the convoluted tubules. Epithelial casts were not found. Calcium-oxalate crystals were always present, and frequently those of free uric acid.

The most interesting feature of this urine is that relating to the probable total absence of traces of serum-albumin, in the face of diminution in bulk of that fluid and indubitable evidence, both rational and physical, of renal disease. Very critical and searching examinations were pursued on all occasions, both on each of the days on which estimations were made of the nitrogenous excretion, as already recorded, and frequently since. Yet at no time have I obtained a reaction by any test which indicates the presence of serum-albumin. The methods employed for testing were very similar to those detailed in my former paper, with the exception that dialysis was often also used. In the application of the tests, the filtered, acid urine, which if turbid from urates was cleared by gentle heat, was first carefully overlaid with picric acid; a second specimen was similarly tested with Millard's solution, and still another with glacial phosphoric acid. Any indication of a contact-ring occurring within a few minutes after application of the tests—in about one-

[1] Its usual high acidity did not render necessary the coincident use of acetic acid with the boiling test, save on two occasions in which a slight phosphatic cloud occurred, dissipated by the use of a few drops of dilute acid.

third of the occasions a slight contact haze or ring was usual to picric acid after the tube stood a few moments, usually, however, only when the urine was greatly concentrated—350 c.c. and under—on which occasion also mucous cylindroids were found to abound in the urine—the urine was treated with acetic acid (5 c.c. of 25 per cent. glacial acid to 15 c.c. of urine), thoroughly agitated, allowed to stand, and then filtered. The urine so treated became slightly cloudy, and subsequently deposited fine flakes of mucin. This mucin-reaction was more apparent when the specimen was first diluted with an equal bulk or more of water, though no large additions were practised with specimens to which it was intended to apply tests for albumin, lest minute traces of the latter present might by too great dilution escape detection even by the delicate tests employed. The acidified specimen was then repeatedly filtered and the excess of acid was neutralized by a concentrated solution of NaHO from a burette. After subsequent filtration and acidification the picric-acid test was once more applied. Note of the amount of acid and alkali added was always taken, and a second control-solution of the urine diluted with distilled water to the bulk of its treated companion was tested for albumin similarly to the other, that it might be known whether the occurrence of the disappearance of this reaction to these was rather in consequence of too great dilution than to separation of a substance coagulable with acetic acid, such as mucin or nucleo-albumin. It was found that invariably with urine so treated, had a previous response occurred to the contact-test with picric acid not dissipated by placing the tube in hot water—and this response on no occasion amounted to more than a slight ring or contact-haze, of tardy appearance[1]—none subsequently occurred when the picric-acid test was applied to the specimen so manipulated, though the reaction was always evident in that of the control diluted with water, as stated. The result was similar when Millard's solution[2] was employed, as it was on a number of occasions, to supplement but not to supplant the picric acid. With metaphosphoric acid, used in the glacial state, had a haze occurred with it, as was frequently the case when the contact of the portion of acid was of greater duration than from three to five minutes,[3] no reaction occurred in the acidified, non-mucinous,

[1] No drugs were administered during the time these tests were made, save a nightly dose of cascara, as stated. Dilutions were always carried after the first few trials up to two and a half times the amount of urine used, so that if 300 c.c. equalled the twenty-four-hour quantity, after dilution the amount would equal a twenty-four-hour specimen of 700 c.c., still but about one-half the average normal quantity. Dilution was rendered necessary to obviate danger of reaction between the excess of sodium acetate formed in neutralizing and the tests for albumin employed, thus obscuring the response of the latter. The addition of an excess of acetic acid was necessary to insure any marked precipitation of mucinous substance in a urine so concentrated—rich in salts, the latter preventing separation of the soluble mucin unless acted upon by a great deal of acid.

[2] This solution does not react so strongly to mucin as other of the delicate tests, such as trichloracetic acid, Tanret's and Sebelein's reagents. In Millard's solution acetic acid exists as a base which has little or no precipitating influence in this direction.

[3] A slight response is common in all non-albuminous concentrated urines with metaphosphoric acid at the end of from three to five minutes. This is probably mucinous. Dr. Leff-

unneutralized specimen. After the addition of soda, though the specimen was subsequently rendered acid at the end of from ten to fifteen minutes, a narrow, opaque ring would develop, quite unlike what is the case with an albuminous response, just above the portion of acid. This was noticed on two occasions in which this test was so applied. It probably indicates, as Dr. Leffmann suggests to me, a reaction between the metaphosphoric acid and the excess of sodium salt present or some substance contributed by the NaHO.

Thorough ebullition of the upper stratum of the undiluted acid urine in a test-tube was always negative as regards even a trace of comparative haze. The result of boiling specimens dialyzed to a specific gravity of 1010 and to 1005, by which dialysis most of the salts and coloring-matter were removed, was likewise negative unless a small amount of a weak acetic-acid solution was first added to the yet acid urine. On several occasions, dialyzing carefully to quite a low gravity, the urine still remained transparent and free from more than a slight haze. On a number of occasions but a trace of haze was present, though brought to a gravity of but from 1002 to 1004. This was especially notable when portions of the twenty-four-hour amount which had equalled 400 c.c. or over were dialyzed. When 1002 was reached (which occurred when dialysis was continued for from sixteen to twenty hours) of samples of the urine representing originally great concentration (about 300 c.c), the specimen was usually too turbid from spontaneous precipitation of mucin for the application of tests. Dialyzed specimens from the daily quantity which had equalled or exceeded 400 c.c., and still remained sufficiently free from cloud for testing, treated with HNO_3 by the contact method,[1] and by heat and acid, gave no reaction. When the dialysis was carried to 1001 to 1002, a few drops of a 2 per cent. acid solution alone, or this plus heat, produced some turbidity; and subsequently, after standing, fine, flaky strings, characteristic of mucin, separated. That this turbidity was due solely to mucin I think there is no doubt. The addition of more acid to a second portion, without the employment of ebullition, allowing also this portion to stand until precipitation of the mucin was assured, prevented more than the slightest contact-haze, when any was apparent to the contact-test with picric acid and with Millard's solution. This same dialyzed urine always responded markedly by the contact-method to citric acid, especially when used in 25 per cent. solution, giving a much broader contact-haze than did picric acid. This was evident, too, in the undiluted, undialyzed urine. Here, in testing with various percentages of

mann, who has for years employed the metaphosphoric acid in albumin-testing in preference to all others, tells me he disregards a response not occurring within three minutes' contact.

[1] After dialysis, by which salts and coloring-matter are removed, the contact HNO_3 test is of far greater delicacy for the detection of traces of serum-albumin than before. See a very interesting and instructive paper by Kirk on mucin-testing in the urine, *Lancet*, April 26 and May 3, 1890.

citric acid, from a saturated solution to a low dilution, it was found that a 33 per cent. solution gave the most marked contact-response. With this percentage the reaction was always quite pronounced. In the dialyzed urine the addition of a few drops of a 25 per cent. citric-acid solution always induced some opacity.

The urine under consideration was especially interesting for study because of the small quantity voided. It represented in amount one already reduced by concentration from a normal 1200 c.c. to an average of 350 c.c. On some days it was considerably less in amount than this, the lowest being 250 c.c. (8½ fluidounces). It would be surprising, indeed, if a urine so concentrated and so rich in cellular elements as this was shown microscopically to be did not respond to the more delicate tests for albumin, because of the concentrated normal amount of nucleo-albumin, or mucin, it must contain, derived from the urinary passages outside the kidneys, disregarding even that no small quantity probably present in this case, as in all of this sort, derived from the renal cells themselves, due to their defective metabolism.[1]

It seems now established that serum-albumin as an exudate from the blood takes no part in the formation of casts. That these may be present without an accompanying albuminuria is certain. This dissociation was well illustrated in the previous series of cases of non-albuminous nephritis that I reported. In the urine of all of these casts were more or less frequently present, as in the present case. Casts are now regarded on the best of evidence as the product of faulty metamorphosis or inflammatory irritation of the renal epithelium,[2] by which a secretion from or a disintegration of the protoplasm of the latter occur, resulting in the formation of cylindroids or true cylinders. With the presence of mucous cylindroids in any amount, should the urine be concentrated, mucin is also present in amount usually detectable by acetic acid when added in excess, and unless regarded will mislead when delicate tests for albumin are employed, as these, without exception, also react to mucin.[3]

[1] As to this see the last portion of the second part of Kirk's paper, before referred to. Kirk there calls attention to a *renal mucinuria* independent of albuminuria and to the frequent association of the two in ordinary nephritis. This last is well known. In my case the persistent low excretion of urinary salts and the presence of casts and cylindroids well indicated the tubular disturbance of function.

[2] See Lubarsch, Centralbl. f. allgem. Path., Band iv., No. 6, and Aufrecht, Centralbl. f. klin. Med., June 30, 1893.

[3] I have left the further consideration of this part of the subject to be taken up in a second paper devoted to fallacies attending the employment of certain delicate tests for serum-albumin, notably the trichloracetic acid.

I think that it may be accepted in the case just detailed, as in those of the others previously reported, in all of which a more or less concentrated urine occurred, that even small amounts of serum-albumin are in all probability absent, and that from the behavior of the urine to picric acid, when a slight reaction with it was obtained, as sometimes occurred, that this was in all probability due to the presence of an albuminoid of a mucinous (or nucleo-albuminous) nature. The urine of several of these cases, especially when much concentrated, was rich in mucin yielding cellular elements derived from the genito-urinary passages, apart from that probably originating from the epithelium of the kidney. In granular kidney, in those cases in which the urine is often singularly free from sediment, and of low gravity, showing diminution in salts, and in which the prior use of acetic acid without heat causes no cloud, but with which the most reliable finer tests for albumin respond, the reaction in all probability is that of serum-albumin. I recently examined the urine of a case of this sort over some days. It had been suspected by the physician to be albumin-free. The patient was senile, with an hypertrophied heart and rigid arteries. The urine was subnormal in amount, of low gravity, low urea, chlorides, and uric acid, and deposited no sediment, even after twenty-four hours' standing. Specimens centrifugalized likewise yielded none. No reaction occurred to Heller's contact-test with HNO_3, but to picric acid and metaphosphoric acid a response was evident, and on two occasions a slight opacity was detected by boiling, with the addition of acetic acid.

LEPROSY.

April 4, 1894.

Dr. Benjamin Sharp, of the Academy of Natural Sciences, gave, by invitation, an exhibition of photographic lantern pictures of the Leper Settlement at Molokai, Sandwich Islands.

Dr. James C. Wilson also exhibited specimens of leprosy from the Trinidad Leper Asylum, sent by Dr. Beaven Rake.

DISCUSSION.

Dr. Louis A. Duhring: This subject is one of great interest and extremely extensive in its ramifications, so that one is at loss to know where to begin to speak; whether to take up the symptomatology, the etiology, or the pathology. They are all extremely interesting. Much as I should like to speak at length on this subject, I shall confine myself to a few pertinent points suggested by the remarks of Dr. Sharp. It was stated that in Molokai the tubercular manifestations predominated over the anæsthetic. This is an interesting fact, for tubercular leprosy is usually more common in northern climates, as Iceland, Norway, and Russia, whereas the anæsthetic form is more common in warm climates. In Norway the proportion of the different varieties is about 55 of the tubercular, 30 of the anæsthetic, and 15 of the mixed form. The photographs presented this evening show a combination of the two chief forms to a marked extent. The coexistence of the tubercular and anæsthetic forms is very properly described as the mixed variety. There is no disease affecting the skin, with the exception of syphilis, which presents such a multiformity of lesions as lepra.

The subject of etiology is one of great interest. The most competent observers in the world, those who have had the largest opportunities for observation, are not in accord as to the contagiousness of the disease. The majority are opposed to the idea of ordinary contagion. Dr. Sharp has alluded to the fact that none of the asylum attendants had contracted the disease. This is in accord with the experience at Trinidad and elsewhere. A search of literature reveals but few cases where the disease has been communicated to physicians or nurses by supposed contagion. A few days ago I was talking in New York with a medical man who sees leprosy occasionally as it passes through that city. He informed me that very little

special care was taken of the lepers in the hospitals to guard against contagion, and that the Board of Health did not assume control of such cases. That the disease is infectious or contagious, we must admit, but the mode of propagation is extremely obscure. The mode of entrance of the bacillus is a point of interest. There is no initial lesion. So insidious is it that it leaves behind no trace of its mode of entrance in the way of induration or sore, which we should expect, because in many respects it follows the natural history of syphilis. We know that in certain places leprosy spreads, and that in other places where it was rife many years ago it is diminishing, and not as a result solely of segregation. In Norway segregation has partly proved a failure, for it is not thoroughly carried out. The disease has a marked tendency to begin in the integument; it seems that in the beginning it is peripheral, and that even in time it only becomes partially central. It moreover probably begins about the peripheral nerves of the skin and mucous membrane. The anatomical investigations of Dr. Gerlach are interesting in this connection. He has shown by dissections that the disease in its incipiency is in the peripheral nerves, and that it works up the nerves to the central organs. He has traced the degeneration of the nerves up toward the trunks. The bacilli are abundantly found in the corium, but comparatively few are seen in the nerves. They are rarely found in the large nerves, cord, and brain, and not often in the large organs.

Dr. Joseph Leidy, Jr.: Some two or three years ago I visited the leper hospitals of Norway and the north of Russia. In Norway I saw between eight and nine hundred cases, and as many more along the Baltic. It is rather interesting to note that it was the experience of Prof. Hansen, of Bergen, that the anæsthetic form seemed to be rather self-limited, that it ran its course and then ceased to progress, while the tubercular and mixed forms were continuous, involving the integument and extending to the mucous membranes, first of the lips, then of the tongue, epiglottis and larynx, producing such ulceration and contraction as necessitated tracheotomy. Finally the disease extended to the lungs, the patients dying of tuberculosis.

With reference to the bacillus, my own experience in examining many sections in Prof. Hansen's laboratory was rather different from that of Dr. Duhring. I did find in the nerve trunks and in the peripheral nerves many bacilli. They were found in the connective tissue corpuscles and not at large.

The treatment there was isolation. By this means the number had been reduced in thirty years from 5000 to 3000. In Finland the disease seemed to be at a standstill.

With reference to the appearance of the disease on the skin, although it has the reputation of being a very loathsome disease, yet I have seen many cases of syphilis of the skin far more repulsive than those presented by the tubercular forms of leprosy.

The form most common along the coast of Norway is the tubercular, while that inland is the anæsthetic form, though the mixed forms are to be found in both localities. Two years ago a patient presented himself at the Infirmary for Nervous Diseases for a curious nervous affection involving the upper extremities. This man said that he had lost sensation in the palms of the hands and the fingers. He also had two or three small sores upon the fingers. It occurred to me that it might be a beginning case of leprosy. On cross-examination he admitted that he had been a barber at Honolulu twenty-two years ago, and had been in this country four or five years. This man was advised to return to his home, which I believe he did. In this case the ulceration upon the fingers was mechanical, as are many of the skin lesions upon the hands in the anæsthetic forms.

Dr. H. W. Stelwagon: This is one of the oldest diseases of which we have knowledge, and has been discussed from time immemorial. This makes it the more strange that there is so little unanimity of opinion among physicians as to its causation. Climate, soil, race, malaria, food, residence by the seashore, heredity, and contagion—each has had its warm supporters. With the exception of food, heredity, and contagion, each of these various alleged causes, while possibly of predisposing influence, has gradually been eliminated as potential factors, as the disease may occur in any climate, although more common in tropical and subtropical countries; it is seen in countries of various conditions of soil, in all races, in non-malarial as well as malarial districts, inland as well as by the sea. It is also met with in those addicted to fish, meat, or mixed diet, although it is by far the most frequent in those classes and races in whose food supply fish and the poorer meats, often of a partially putrescent character, form an important part. Heredity is still actively discussed as the prime etiological element, but if the facts apparently substantiating this claim are carefully sifted and followed up, they will be found not only inconclusive, but will admit of other explanation, and the opinion is gradually pointing toward this as an influential factor only in the sense that the predisposition or susceptibility to the disease is transmitted.

Contagion has always been discussed as a cause of the disease, and in certain countries it has been maintained throughout that the disease is contagious. In other leprous districts there is complete disbelief in the communicability of the disease. Since, however, Hansen, in 1874, discovered the bacillus the contagion theory has been constantly gaining ground, although both before and since subjected to powerful assaults. Thus the commission appointed by the Royal College of Physicians of London, in 1867, reached the conclusion that leprosy was not contagious. A second commission appointed a few years ago came, practically, I believe, to the same conclusion, although a strong minority maintained that the disease was contagious.

The inoculation of an Hawaiian criminal has been mentioned. This was

done successfully, but Crocker states, in referring to this experiment in his treatise, that in this man's family there subsequently developed several cases of leprosy, and it is presumable that it might have existed in a latent form in the criminal himself. This case, therefore, does not have the force that it at first seemed to have. In my judgment the contagion theory explains the experiences and observations recorded of the disease better than any other theory so far advanced. There are, it is true, certain facts which seem to bear against it. Lepers have lived with unaffected wives, and the reverse, and the healthy individuals have not become affected. Physicians and nurses brought in close contact have only exceptionally contracted the disease. The children of lepers frequently remain free from any manifestation of the disease. The case is reported of a Norwegian leper in Minnesota, the father of children, grandchildren, and great-grandchildren, many coming in close family contact with him, and yet none have become affected. While admitting that the disease is contagious, doubtless only by inoculation, it must be assumed that there are certain unknown conditions bringing about in those exposed to the disease a receptivity; in other words, that it is contagious only under conditions not yet understood.

[Dr. Stelwagon also exhibited photographs of lepers coming under his observation.]

Dr. Richard A. Cleemann: I would mention a case simulating leprosy, where the initial lesion was a Colles fracture. The patient was an Irish woman living in Philadelphia. She broke her radius, and after union had occurred she suffered with neuritis, which became ascending. The hand became claw-shaped and the face leonine in expression. There was sclerosis of the skin under both eyes and the lips became so hard that it was with difficulty that she could eat anything. The body became covered with tubercles about the size of an almond. This woman died of the disease, apparently the result of exhaustion. It looked more like the cases of leprosy that I have seen than anything else.

When I was in Norway I visited some of the hospitals and was informed by the nurses that they took no special precautions and that they knew of no one contracting the disease. They did not seem to be very strict in Norway about putting the lepers in hospitals. I discovered that a man who offered himself as my guide was suffering with leprosy.

Dr. J. C. Wilson: In a recent report of the Trinidad Asylum, which Dr. Rake forwarded to me, there are mentioned two or three facts of great interest. One relates to the etiology. Dr. Rake appears to entertain the view that leprosy is not directly contagious, but that the infecting prineiple may occupy a position outside of the body in the form of spores. Dr. Rake suggests that the question of the contagiousness of the disease cannot be settled until an individual is inoculated in a country where the disease is not endemic.

Another point of interest is that a large proportion of the cases of leprosy in the Trinidad Asylum die of tuberculosis, chiefly of the lungs.

A third point is that wounds in lepers heal with great readiness and that surgical interference has given relief to conditions previously considered irremediable. In speaking of the management of perforating ulcers of the feet, he states that where he has resorted to free incision he has, in many cases, effected a cure.

Dr. Charles W. Dulles: I was much interested in Dr. Sharp's statement with reference to the peculiar shortening of the fingers, with integrity of the skin, and retention of the nails. This would indicate the occurrence of atrophy of the bones. It is well known that leprosy affects in its progress the bones as well as the other tissues. I have recently been investigating some questions in regard to the bones, and I should be glad to hear remarks from those familiar with the subject on the pathological processes of leprosy in the bones, and what is the process that results in shortening of the phalanges without destruction of the skin.

Dr. Arthur Van Harlingen: We have had so many observations with reference to the clinical features of leprosy, that its objective appearance is now pretty well known. A great number of papers have been published in the last few years, giving enormous statistics tending to prove the contagiousness of the disease, and on the other hand, a number of papers proving the non-contagiousness of leprosy have also been published, so that it is difficult even to keep up with the literature of the subject, much less to form any conclusion as to the real facts in the case. My impression is that the direction which investigations should now take is in regard to the life-history of the bacillus. There is no question but that the bacillus of leprosy is the cause of the conditions which we find. The points that remain for investigation are how does the bacillus enter the body, and does it enter in the shape in which it is found. Dr. Wilson has referred to investigations going to show that the bacillus may take on some intermediate form. The idea of an intermediate host has also been suggested. Bevan Rake has pointed out that in all probability the disease is inoculated through the skin. Some of the early observers endeavored to inoculate through the blood, but without result. Some clinical observers have reported vaccination as a cause of leprosy. Many of the cases reported as showing contagion are more than questionable. Several cases are reported of transmission of leprosy, occurring in countries where the disease is not endemic. A man with leprosy in coming from India died and left his clothing to a brother in Ireland, who subsequently developed the disease. But criticism has since thrown doubts upon this case.

As I have said, the study of the disease from a clinical standpoint has gone as far as possible. I think that in this city we have had advantages for studying this affection which have not been properly utilized. The bacillus has been found in almost every tissue, but its mode of development

has not been studied. It has recently been observed that the periphery of the nerve may be the seat of infection.

Looking over the recent literature of leprosy, I have come across one observation which bears upon the manner in which the bacillus may be introduced. In one case it has been in and found around the sebaceous and sweat glands.

I wish again to impress the necessity of studying the life-history of this bacillus. It is cultivated with great difficulty. It is difficult to inoculate. Recently, one observer has examined a mosquito, with the idea that the mosquito might be an intermediate host. Elephantiasis Arabum, we are pretty sure, is due to infection carried by the mosquito. The search for an intermediate host may lead to a solution of the problem and may be suggested as a direction in which these investigations may profitably be carried on.

Dr. Leidy: No matter what the etiology of leprosy, the consensus of opinion among the investigators in the north of Europe is that it requires long contact to communicate the disease. If this is so it is of importance to us and to the patient himself. These remarks are suggested by a case which I saw with Dr. Lewis and which Dr. Duhring also saw. This patient had lived for many years in various cities without any one becoming infected. It would have been a rather severe measure to have confined him. In the present state of our knowledge, admitting that it requires a long contact, the Board of Health should adopt some rule in regard to certain contagious diseases such as leprosy.

Dr. Duhring: With regard to the length of time required for infection, I would say that an officer of the British Army came under my observation while in London. He had gone to India healthy, remained there four or five months and came back a leper. He had seen lepers in the streets, but never came in contact with one. There are other cases reported where persons have contracted leprosy by travelling or sojourning a short time in leprous countries. It would therefore seem possible to contract the disease without coming in direct contact with the leper.

Dr. A. A. Eshner: There would be more harmony in our discussions if, instead of using the word "contagious" in connection with such diseases as leprosy and tuberculosis, for instance, we should speak of these as *transmissible* diseases. Admitting each to be dependent upon a finite cause—a vegetable microörganism, it is easy to understand how the propagation of both diseases is favored by the association, particularly the intimate association, of the well and the sick. With reference to the question of long contact, it may be that such contact is not essential to infection, but that the period of incubation is long. Infection may take place after contact of variable duration, but the outbreak of the symptoms may be long deferred.

Dr. G. G. Davis: I should like to present one practical aspect of this subject derived from a personal experience. While on duty in the Surgical

Dispensary of the Episcopal Hospital, and having charge of diseases of the skin, I noticed among the patients in the waiting-room one with the so-called leonine face. Although I had never seen a case of leprosy, the presence of the affection was at once suggested, and an examination of the body revealed the dark mottled stains, thickened skin, etc. I hesitated to say anything about the case that would betray its character, for I was certain it would create a great commotion. If I happened to be right, the man was doomed, for the case would come before the Board of Health, and he would be sent to the Municipal Hospital and isolated from the community—a most terrible fate. If I should be wrong, what an exceedingly disagreeable predicament I would be placed in? I chose a sort of a middle course, and giving the patient a note, referred him to a friend in whose judgment I had confidence. He confirmed the diagnosis, and kindly relieved me of the disposal of the patient, and I was not called upon to pursue any further measures.

Dr. W. M. Welch: I wish merely to refer to two matters suggested by the discussion: First, I would state that every case of leprosy which has been received into the Municipal Hospital has been examined for the lepra bacilli, and these bacilli have been reported as present. Secondly, it has been said that the period of contact must be long in order for infection to occur. There may be some truth in that; but it is well known that the period of incubation is a long and variable one. A case is on record in which an account is given of two boys playing together—one was a negro and a leper and the other a white boy. The negro was in the habit of amusing the white boy by showing him how far he could run the blade of his penknife into his skin, where doubtless there was an anæsthetic patch. The white boy, not to be outdone in bravery, took the penknife and did the same thing in his own flesh. Nineteen years afterward he developed leprosy.

[Dr. Welch exhibited some photographs of lepers who had been in the Municipal Hospital.]

ACUTE APPENDICITIS.

By JOHN B. DEAVER, M.D.,

[Read May 2, 1894.]

History.—More than sixty years ago Albers, of Bonn, first described, under the name of typhlitis, localized inflammations about the cæcum, of four varieties:

1. Stercoral typhlitis—stagnation of fecal matter in the cæcum, with subsequent irritation.

2. Simple typhlitis—catarrhal inflammation due to a multitude of causes.

3. Perityphlitis—extension of the inflammation of the mucous membrane to the external coat of the cæcum and the surrounding parts.

4. Chronic typhlitis.

Previous to 1820 Dupuytren recorded observations calling attention to the connection between abscess of the right iliac fossa and disease of the cæcum. In 1827, Husson and Dance described the disease more in detail. In 1824, Louyer and Villermay gave the first description of a fatal peritonitis due to perforation of the appendix. Melier, in 1827, reported four cases, three of which were of perforative appendicitis with fulminating peritonitis; the other being one of relapsing appendicitis. He laid especial stress upon two distinct symptoms in the perforative cases, namely, more or less severe abdominal colic, and fixed pain localized in the right iliac fossa. Melier not only described the causes, character, and consequences of appendicitis, but foresaw the possible advantages of operation in this affection. He said: "If it were possible to establish

with certainty the diagnosis of this affection, we can see the possibility of curing the patient by an operation. We shall, perhaps, some day arrive at this result."

Subsequent memoirs of Ménière and Albers, advancing the notion that the lesions occupied the cellular tissues of the right iliac fossa, caused Melier's theories to be lost sight of. Later memoirs of Bodart, Favre, Forget, and of Leudet, added new facts to those established by Villermay and Melier.

Thus little by little the view that the grave and fatal forms of typhlitis are due to perforation of the appendix gained ground, and that the benign and curable forms are due to an inflammation of the cæcum and the cellular tissues which surround it. The theory still counts a few advocates, but fortunately they each year grow fewer and fewer. This much-vexed question has been restored to the status to which Melier had advanced it by the surgeons and physicians of the United States. They have demonstrated by early operation that inflammation of the right iliac fossa is invariably due, primarily, to disease of the appendix vermiformis.

Hancock, in 1848, operated upon one case, and advocated early operation. This met with no encouragement. In 1867, Willard Parker, of New York, first proved that early operation would save 75 per cent. of the cases. Dr. R F. Noyes, in 1883, reported 100 operations, 90 per cent. of which had been performed in America.

An article in the *American Journal of the Medical Sciences*, by Reginald Fitz, published in 1886, "On Perforative Inflammation of the Vermiform Appendix," marks the epoch of the present status of this much-discussed trouble. Fitz collected 209 cases of typhlitis and perityphlitis, and 257 cases of perforative appendicitis. His conclusions have been summarized by Talamon as follows: "He showed that the symptoms are the same in the latter as in the former class of cases. He studied with care the consequences of perforation. He established the fact that the peritonitis is not always generalized, but that it may be circumscribed under the form of an encysted purulent collection. He gave the characters of the tumor

formed by this localized peritonitis, the different modes of the evacuation of the pus, and the complications that may supervene if the disease is left to itself. He insisted on the frequency of fecal concretions as a cause of perforation of the appendix. He concludes in favor of early surgical interference."

In a second paper published by Fitz, in 1888, he advanced the radical but sound theory that the diseases described as typhlitis, perityphlitis, paratyphlitis, appendicular peritonitis, and perityphlitic abscess, are all varieties of one and the same affection, namely, appendicitis.

Anatomy.—The vermiform appendix is a narrow musculo-membranous tube ending in a blind extremity, and is the atrophied remnant of the elongated cæcum. It is present in man and in some of the higher apes only. The elongated cæcum persists in other mammalia. It is attached to the inner and posterior wall of the cæcum, the point which originally was its apex.

The appendix may arise from the apex of the cæcum and be a direct linear continuation of the colon, thus affording an example of the fœtal type. It may arise from the lower end of the cæcum, under which circumstances it is unequally divided by the anterior longitudinal band, that portion to the right of the band being larger, while that to the left, though smaller, constitutes the true apex of the cæcum. This is the type of cæcum usually found when there is an increased development of the anterior wall, which rotates the apex posteriorly and to the left, thus bringing it into close relation with the ileo-cæcal junction. Finally, it may arise from the lower end of the cæcum, appearing between two bulging sacculi and posterior to the inferior angle of junction of the ileum with the cæcum.

In cases of non-descent of the cæcum the appendix will hold a corresponding abnormal position, when it may lie to the left of the median line. Lennander mentions the case of a boy of sixteen, in which the cæcum and the appendix, which was nine inches long, were found lying against the spleen in the left hypochondriac region.

The length of the appendix varies from one to nine and a

half inches, the average being four inches. When the appendix is long, the cæcum as a rule is small. Its diameter is that of a goose-quill or about as thick as a large earthworm (Holden). The appendix is hollow and has an opening of communication with the cæcum, which is usually guarded by a valve of mucosa.

The left or under layer of the mesentery joins with the serous covering of the appendix, which may be complete or incomplete, forming the mesentery of the appendix or the meso-appendix. In the majority of instances the appendix has a peritoneal covering throughout its entirety. When only a portion of the appendix is included in a peritoneal investment, it is the base or root that is destitute. The root of the appendix being uncovered makes this part of the organ extra-peritoneal. If perforation should take place with this disposition of peritoneum, we can clearly see how infection of the post-peritoneal connective tissue would occur and result in the formation of an abscess which would be extra-peritoneal. Perforation of the appendix between the layers of the meso-appendix would cause infection of the post-peritoneal connective tissue.

The meso-appendix is triangular in shape, the base corresponding to the free margin, and the sides to the appendix and the mesentery. It is attached to the proximal one-third or two-thirds of the appendix, leaving the tip free, or it may be attached along its entire length, as I have usually found it. Sometimes it may be altogether absent or exist in so slight a degree as to be practically absent.

It consists of two layers of peritoneum between which run the appendicular artery and vein, and the lymphatics, in addition to some fat. Occasionally the meso-appendix contains a considerable amount of fat which, when operating for its removal, renders its more liable to be torn in the delivery of the appendix. The mobility of the appendix depends upon the extent of the attachment and the width of the meso-appendix.

The meso-appendix may have a hole in it through which the small intestine has been known to herniate and become stran-

gulated. The iliac vessels have been known to pass through the layers of the meso-appendix. In the female it has a prolongation which is lost in the broad ligament; this is described by Clado as the appendiculo-ovarian ligament.

The shape of the appendix varies in accordance with the length of the meso-appendix. When it is short the appendix will present an abrupt curve. If the meso-appendix is shortened in several places it may present a corkscrew shape.

The appendix will occupy one of eight positions in the majority of all cases. Dr. Bristow suggests a very simple method of classification of the positions and directions of the appendix. It consists in locating a central point in the right iliac fossa which represents the attachment of the appendix in its most common position. From this central point are drawn lines radiating in eight different directions. Fowler has modified this method by substituting the initial letters of the points of the compass for the numbers. The central point is located by drawing a line from the anterior superior spine of the ilium to the umbilicus. A point on this line from two to two and a half inches within the anterior spine will correspond to this central point.

The appendix may occupy either of the ileo-cæcal fossæ, more likely the inferior or the sub-cæcal fossa. In the event of its occupying the two latter fossæ, it would sometimes constitute a retroperitoneal hernia of the appendix. Again, if it should occupy one of these fossæ and the mouth of the fossa close over it, it might be regarded by the operator as being absent. Suppuration due to inflammation of the appendix so walled in would be circumscribed. Other abnormal positions of the appendix are met with, namely: (*a*) when it lies behind the peritoneum, below the cæcum and adherent to its under surface, in contact with its muscular wall, and covered by the peritoneal coat of the cæcum; (*b*) adherent to the peritoneum along the right border of the cæcum and ascending colon; or (*c*) adherent to the peritoneum at any point in the neighborhood of the cæcum; and (*d*) in the inguinal canal.

When the appendix lies post-cæcal and post-colic, suppura-

tion, the result of appendicitis, may simulate a lumbar or a perinephritic abscess.

The normal positions of the appendix are five:

1. When it lies under the inferior layer of the mesentery directed toward the spleen.

2. When it lies on the ileo-pectineal line or projects into the pelvis. In this position it may become adherent to the bladder. Only in this position is rectal examination of value.

3. When there is a long meso-appendix, it may lie to the right of the cæcum and the ascending colon, running upward parallel to the colon over the kidney and toward the right lobe of the liver.

4. When it lies in front of the colon and cæcum.

5. When it lies under the cæcum.

Usually the width of the meso-appendix accounts for the other three positions by allowing more motion to the organ.

Etiology.—The theory that appendicitis is due to a foreign body in the appendix—for example, seeds, pits of the smaller fruits, pins, buttons, etc.—has been accepted etiologically in a certain proportion of cases, and fecal concretions for the balance. Recent writers upon this subject have taken exception to this theory, basing their arguments on the fact that cases of appendicitis, with or without perforation, have occurred in which no foreign body could be found. They also present cases in which normal appendices have been found, while performing abdominal section for other conditions, as well as in post-mortem reports, in both of which fecal concretions or inspissated mucus was contained within the appendix without evidences of inflammation, and when there had been no history of symptoms of appendicitis. In every case of appendicitis these observers have demonstrated the presence of the bacterium coli commune in pure cultures or associated with pyogenic organisms. They have grown pure cultures of this bacterium from the walls of an inflamed appendix in which perforation had not occurred, and have also grown pure cultures from the interior of the appendix, and from the pus of an appendicular abscess. This bacterium is normally found

within the colon and the appendix; still it has been proved that under favorable conditions—as, for example, inflammation of the appendix—it takes on pathogenic and pyogenic properties. The bacterium coli commune has been grown in pure cultures, or associated with other pyogenic micro-organisms, from the exudate of peritonitis, from the pus of a septic pneumonia following a perforative general peritonitis, and also from the spleen and liver of such cases.

From the number of cases in which I have found foreign bodies, it is my judgment that in the majority of cases of perforative appendicitis there is a direct relation between the presence of the concretion and the other causative agents. From the fact that so many cases of appendicitis follow so closely upon the ingestion of improperly masticated and hastily swallowed indigestible food, I cannot help thinking that there is a direct relation of cause and effect between them.

I would classify the causes of appendicitis under two heads—predisposing and exciting.

Predisposing Causes.—*Age.* 15 per cent. of all cases occur under fifteen years of age, but the disease is most common between the ages of ten and thirty. The earliest age in which I have met with the disease was in a child under two years.

Sex. Males are more prone to the disease than females.

Constipation, from its tendency to provoke catarrhal inflammation.

Tuberculosis, when secondary to the involvement of the cæcum. Should the disease be most pronounced in the appendix, it is likely to be followed by a result similar to that of acute perforative appendicitis. The appendix being a rudimentary organ, and therefore of lower vitality than the cæcum, is more likely to suffer from pathological processes.

By far the most frequent cause is a *previous attack*, a history of which cannot always be obtained on account of its mild character.

Exposure to cold and wet. I have met with two cases illustrating these agencies. In one the attack was provoked by

taking a cold shower-bath after coming out of a warm bath; the other was the result of wet feet.

Traumatism, such as from violent exercise or sudden blows. The former is more likely to cause the disease if it immediately follows the taking of a heavy meal. As I have already stated, the rapid ingestion of indigestible food is a cause.

Exciting Causes.—*The introduction of foreign substances*, such as fecal matter, pits, or enteroliths, when associated with the ever-present bacterium coli commune, is the most frequent exciting cause of appendicitis.

Pathology.—In considering the pathology of acute appendicitis there is a great difference of opinion as to the cause in localized abscesses of the peculiar offensive odor which the pus possesses; whether this odor is due to a commingling of the gases of the bowel which are intimately admixed with the pus, or whether it is the result of decomposition of the contents of the abscess. I believe this odor to be entirely fecal, as we find that abscesses occurring elsewhere in connection with the bowel will possess the same characteristic odor which is found in the appendiceal abscess.

Cases which take on the gangrenous type of inflammation may lead to the destruction of the cæcum at the base of the appendix. I have seen several such cases during the past year.

Tuberculosis has been found in my experience to be one of the remote causes of suppurative appendicitis.

The rôle which the bacterium coli commune plays in the production of peritonitis lies in the fact that the appendix is a hatching-bed for this dangerous form of bacterium. It can rest within the cavity of the appendix and multiply with perfect impunity, not being in the direct channel of fecal circulation, and irrespective of the fact that intestinal antiseptics may have been administered.

The appendix in an acute inflammatory condition is enlarged, congested, and hard; its peritoneal coat is thickened and at times covered with flakes of lymph. The mucous lining is habitually thickened, not infrequently being almost equal to

all of the other coats. Within it presents corrugations, at times irregular ulcerated areas, and in subacute cases may rarely be the seat of small mucous cysts. If perforation has not already taken place there may be seen a dark spot of softening, containing within its tissues colonies of the bacterium coli commune. If perforation has taken place, the tissue surrounding the opening of communication will extrude through it.

In perforation of an appendix in which there is a fecal concretion, the perforation usually takes place at the situation of the concretion. There may be gangrene of the entire walls of the organ, leaving a direct communication with the bowel; or only of the outer coat, leaving the mucous membrane intact. If the appendix be turned upon itself and its inflamed end be in contact with the cæcum, ulceration at the spot of contact will occur. I have recently operated upon a case in which such a condition of affairs was present.

On several occasions, while washing out the abscess-cavity I have seen a gangrenous appendix that had ulcerated off appear in the wound.[1]

Clinical History.—Acute appendicitis is usually ushered in suddenly by a severe pain which may be peri-umbilical or epigastric. The character of the pain is paroxysmal and colicky, varying in intensity. Coincident with the pain there is vomiting, first of the contents of the stomach, later of bile-stained material. The colic now subsides, and the pain shifts to the right iliac fossa and becomes constant. There may be a point of greatest intensity, or it may be diffused. The abdominal muscles of the right side are contracted and rigid. There is but little fever. The attack may terminate here with a gradual subsidence of all the symptoms; or on the other hand, this attack may be followed directly by a return of the abdominal pain, and vomiting which is incessant and uncontrollable. The entire belly-wall becomes tense and rigid, though the more so on the right side. The patient takes on the typical and anxious expression of the Hippocratic face, and

[1] Microscopic and bacteriologic examinations are being made by Drs. Stengel, Abbott, and Kneass, and will be published at a later date.

symptoms of collapse appear, with sweating, and cold and blue extremities, death finally supervening from diffused septic peritonitis.

In another type, the pain, which at first has been diffused, concentrates itself in the right iliac fossa. The accompanying rigidity of the muscles of the right side is replaced by a swelling which in some instances presents to the touch a doughy sensation, and in others that of a hard mass the contour of which is not constant.

The fever, which up to this time has been of a moderate degree becomes pronounced, with a decided rise in the evening and remission in the morning. While this symptom usually indicates pus, the absence of a rise of temperature does not necessarily preclude the possibilities of the presence of pus. This I have demonstrated many times by operation to the skeptical attending physician, who has pinned his faith to the thermometer, which instrument is of but little value in the majority of this class of cases.

Symptoms.—*Pain.* The initial symptom is pain. This usually follows the ingestion of a hearty meal of indigestible foods, improperly masticated and hastily swallowed, particularly when eaten at night. The character of the pain at the outset is paroxysmal and colicky. So far it simulates an attack of acute intestinal indigestion. The term appendicular colic has been applied to this initiatory pain. To this, however, objection has been raised, as the cause of pain is inflammatory and not functional. While acknowledging the cause of the pain to be inflammatory, yet I am convinced that it may be paroxysmal in character; otherwise how can we explain the wave-like exacerbations so vividly described by an intelligent patient. Palpation over the affected area excites and intensifies this peculiar, wave-like, characteristic pain. Fowler, who in a recent article upon appendicitis has taken exception to the term appendicular colic, offers as an argument the absence of poor development of the circular muscular fibres of the appendix, and from this argues the inability of the appendix to cause this colicky pain by expulsive efforts. I must take exception

to Fowler's statement, particularly regarding the absence of circular muscular fibres in the appendix, as this organ possesses a continuous circular muscular coat. I will, however, grant that the circular muscular fibres are not always as well developed as the longitudinal, but I am convinced that the appendix possesses sufficient contractile power to cause a colicky pain by its expulsive efforts. The study of pathological appendices cannot be relied on for histological facts. The dissection of normal appendices proves the presence of circular muscular fibres. The irritation arising from a simple catarrhal inflammation of an appendix in which there is no fecal concretion or foreign body, is capable of giving rise to expulsive efforts which cause the colicky pain and which may be likened to similar efforts of an inflamed rectum or bladder.

Location of pain. The primary pain is referred most frequently to the umbilicus or peri-umbilical region; next in order of frequency to the epigastrium; and last of all to the region of the appendix. After the occurrence of localized peritonitis in the right iliac fossa the pain is here located. I have observed two cases in which the pain was referred to the left iliac fossa, in neither of which, as demonstrated by operation, did the appendix occupy the corresponding position. In one the appendix hung down into the pelvis and in the other it was retro-cæcal.

Rigidity of the abdominal walls. This, next to pain, is one of the most reliable signs. It is usually confined to the right side of the abdomen, being most marked over the inflamed region, and immediately follows the localization of pain in this locality. In some instances the rigidity is so pronounced that it operates against deep palpation, and in addition makes the percussion-note of higher pitch.

Vomiting. Coincident with the onset of the initial pain there is vomiting of the contents of the stomach. This usually does not persist in favorable cases, while in unfavorable cases it is persistent and uncontrollable. The ejecta consist first of the contents of the stomach, later of bile, and finally, if intestinal paresis supervenes, consequent upon a septic peritonitis, of ster-

coraceous matter. When the material rejected becomes stercoraceous, it is thrown off by regurgitation, indicative of a fatal termination.

Constipation. Constipation is present in the majority of cases of appendicitis, yet in a few diarrhœa ushers in the attack. Obstinate constipation early in the disease is due either to intestinal paresis, the result of infection, or to the indiscriminate use of opium.

The temperature and the pulse-rate bear no direct relation to the gravity of the attack. We may have early perforation and gangrene of the appendix with but a moderate rise of temperature; on the other hand, there may be a decided rise with a simple catarrhal inflammation of the appendix. The pulse in the former class of cases more nearly corresponds to the gravity of the attack. A sudden fall of temperature to the normal or below normal is not by any means ground for a favorable outlook, as too often it indicates the lull which immediately precedes the storm of destruction in the shape of perforation or a ruptured abscess. This is in accord with the statement that I have already made, namely, that the thermometer is a most untrustworthy instrument in determining the severity of a case.

Tenderness is one of the most valuable and constant of all the signs or symptoms. It is always present, although if the appendix be post-cæcal and the rigidity of the abdominal walls marked, it is difficult to elicit, requiring deep palpation. Sometimes it can be elicited through the rectum or vagina. In vaginal or rectal palpation in the female, the possibility of a right-sided pyosalpinx and salpingitis must be borne in mind. In rare cases the tender spot may be located in the loin, only to be discovered by deep palpation. After the advent of suppuration the tenderness becomes general in the right iliac fossa and loses its extreme severity. Frequently after the remission which follows the sudden sharp primary attack, tenderness and rigidity alone remain to tell the attending physician that trouble still exists in the right iliac fossa. The point of greatest intensity is usually over the inflamed appendix, but to this

rule there are exceptions. I have recently met with a case in a young adult male, in which the point of greatest tenderness was to the left of the left rectus muscle, a little above the level of the anterior superior spine of the ilium. By rectal examination a small but very sensitive mass occupying the recto-vesical space was detected. The operation demonstrated the appendix occupying this position. The point of greatest intensity usually corresponds to the so-called McBurney's point.

I recall two cases in which the point of greatest tenderness was immediately above the middle of Poupart's ligament. This, as demonstrated by the operation, corresponded to the angle of curve in the appendix; in both cases the origin was from the postero-external aspect of the base of the cæcum, descending in front of the latter as far as the apex, where it curved abruptly upward.

Distention may be due to several causes: to mechanical obstruction, to paralysis of the intestines, to septic causes, and to obstinate constipation with consequent collection of gas. Richardson points out the possible differential diagnosis by means of auscultation between distention due to accumulated gas from mechanical causes, and that to paralysis of the intestines, the result of infection—the sounds of peristaltic action being clearly heard in the former condition but not in the latter. The distention may sometimes be limited to the right side of the abdomen; here only those portions of the gut in relation with the inflamed area are affected. This local distention may be marked, because the still functionally active intestine will force more gas into the affected portion. If the peritonitis become diffused, the abdomen is flat and its walls rigid and hard. This appears early and arises from the complete paralysis of the intestinal canal preventing the entrance of gas. A distended abdomen associated with marked and uncontrollable vomiting is an ominous combination of symptoms.

Tongue. The tongue is furred, and if diffused peritonitis occur may become dry, associated with a deposit of sordes upon the teeth.

Urine and bladder. The urine is usually diminished in amount, and contains albumin and indican. Several theories have been advanced to explain this diminished and albuminous urine. The most tenable of these is that there is a decreased activity of the glomerules of the kidney, due to the general fall in the arterial tension. Frequency of urination is often a prominent symptom from the first, being most probably due to disturbance of the sympathetic nerves, and when the inflamed appendix occupies the pelvis, to irritation of the bladder directly communicated thereto. In peritonitis involving the serous coat of the bladder, retention of urine may occur, necessitating the use of the catheter.

Respiration. Respiration plays a comparatively unimportant part in the symptomatology of appendicitis, yet I have noticed early in the attack a voluntary limitation in breathing, the patient thus favoring the tender side. If the distention is pronounced the respiration is correspondingly labored, while if there is active peritonitis the respiration is thoracic. In advanced cases of appendicitis with a diffuse peritonitis, a symptom that I have also noticed is a peculiar reflex condition of the pharynx, which the patient speaks of as a difficulty in swallowing.

Leukocytosis. According to Richardson, leukocytosis is an invariable symptom in perforative appendicitis. In one case he deferred operation for twenty-four hours on account of the absence of this symptom, the patient dying a few hours after the draining of an extensive general infective peritonitis.

Diagnosis.—Ordinarily, I regard the diagnosis of appendicitis as simple. The suddenness of the attack, the pain referred either to the peri-umbilical region or right iliac fossa, the occurrence of nausea and vomiting coincident with the pain, and tenderness over the site of the appendix, are pathognomonic. One of the greatest sources of error is the masking of the symptoms by the use of large doses of opium in some form.

The ushering in of an acute attack of appendicitis simulates very closely that of an attack of bilious colic, the pain being

peri-umbilical or epigastric, and associated with vomiting. Shortly the pain becomes localized and more intense in the iliac fossa, when we should at once suspect more serious trouble. Owing to the fact that appendicitis follows some hours after the ingestion of food, the vomited matter consists of partly digested material, and is often bile-stained. The pain and tenderness cause the patient to voluntarily assume the dorsal decubitus with thoracic respiration, to favor the affected side. Palpation reveals decided rigidity of the abdominal walls, most marked on the right side, and particularly over the right iliac fossa. While it may be slight, yet the experienced sense of touch cannot fail to recognize it. Tenderness is a most important and reliable diagnostic sign. It is always present, and corresponds to the point of greatest intensity of the inflammation, which is the appendix. This may correspond to McBurney's point, or when the appendix holds an anomalous position its location will be governed by that of the organ—as, for example, when the appendix occupies the pelvis, when the point of greatest tenderness is elicited by rectal palpation. Between the disease and the extent and intensity of the tenderness there is a close relationship. When the tenderness continues to increase it shows the progressive tendency of the attack; while if it decrease without the administration of opium, it is indicative of a favorable termination. I have recently operated upon a case in which the greatest tenderness was elicited by rectal examination, and where there was also a distinct point of tenderness to the outer side of the left rectus muscle: this was explained by the presence of adhesions, indicative of Nature's effort to circumscribe the purulent collection.

Fulness. Fulness in the right iliac fossa appears late, after inflammatory exudate and adhesions are formed; coupled with this a doughy feeling may be present, indicating the presence of pus, though this is not a reliable diagnostic sign. Accompanying fulness there may be œdema of the abdominal walls. Excessive circumscribed tenderness I have come to regard as a more reliable sign of the presence of pus than either doughiness or œdema.

When the appendix lies over the psoas magnus or iliacus muscle, flexion of the thigh may be an early sign. Another cause for flexion of the thigh upon the abdomen is rigidity of the anterior abdominal wall due to peritonitis, this, however, being a later manifestation.

Distention. In simple appendicitis the distention, if at all present and not due to mechanical causes, is confined to the seat of the inflammation. Distention occurring in connection with appendicitis is due to peritonitis, and its extent corresponds to the area of inflamed peritoneum. Mechanical obstruction due to constipation, or the use of opium, causes distention, which differs from that of septic peritonitis in being functional, and can be differentiated by auscultation. Functional distention simply occasions discomfort, while that of septic paralysis is indicative of a grave type of the disease. Distention may also be due to acute mechanical intestinal obstruction, such as bands. In some early cases of perforative appendicitis in which the general peritoneal cavity is not yet walled off, and septic peritonitis supervenes, we may not only not have distention, but instead may have a scaphoid or retracted belly, due to profound paresis of the intestinal canal, which would prevent the admission of gas.

Only in cases of appendicitis with circumscribed peritonitis are we likely to detect the presence of a mass. Even under these circumstances rigidity of the overlying abdominal walls may mask this sign, unless it occupy the pelvis, when it can be detected by rectal or vaginal examination. If the patient be a female, right-sided pyosalpinx or salpingitis must be excluded. Rectal examination should be confined to the use of the finger. The introduction of the hand, I maintain, is unjustifiable, as it endangers adhesions and the rupturing of abscesses. Examination under ether will not throw sufficient additional light on the diagnosis of an obscure case to warrant the administration of the anæsthetic.

The advent of suppuration may or may not be accompanied by a chill.

The temperature in appendicitis is so variable and inconstant

that it is of little value except in the differential diagnosis between it and some other conditions in which it is characteristic.

Percussion over the diseased area will give a flat or resonant note depending upon the appendix.

In a doubtful case in which it would seem to be impossible to differentiate, rather than introduce an exploring or aspirating needle, either of which procedures under these circumstances I regard as most unsurgical and attended by no little danger, I would advise an exploratory incision.

Differential diagnosis. Appendicitis may be confounded with the following conditions:

1. *Bilious colic or acute indigestion.* The onset of the attack of appendicitis is somewhat similar to that of an attack of acute indigestion, differing, however, in the character of the vomited matter, and in the fact that the pain soon becomes located in the right iliac fossa, and is accompanied by tenderness.

2. *Hepatic colic.* In hepatic colic the painful area is usually higher in the abdomen, referred also to the shoulder, and most severe over the region of the gall-bladder, and usually followed by jaundice.

3. *Floating kidney.* From floating kidney with a twisted pedicle, appendicitis may be diagnosticated by the presence of blood in the urine, a history of a movable tumor prior to the attack, a depression in the right loin corresponding to the site of the kidney, and possible symptoms of uræmia.

4. *Intestinal obstruction.* In intestinal obstruction the onset is more abrupt, and the pain, which at first was remissive, is referred to the seat of the obstruction, more commonly to the umbilicus; there is absolute constipation and inability to pass flatus, and vomiting occurs early and soon becomes fecal. With the onset of peritonitis regurgitant vomiting occurs, and there is absence of fever unless the patient survive long enough to allow inflammation to set in. Invagination is the most common form of obstruction in children, while obstruction from bands and volvulus are more common in adult life. Tumors from these forms of obstruction are more common to

the left of the linea alba. When obstruction is the result of invagination, blood and mucus will be discharged from the rectum, and upon examination through this avenue a tumor may be felt. The early development of peritonitis in acute intestinal obstruction is marked by great distention of the abdomen. Shock and collapse appear early in the obstruction, but not in appendicitis, unless it be of the fulminating type, and even then collapse does not occur as early.

5. *Pyonephrosis.* From abscess of the kidney appendicitis differs, first, in that the pain in the former radiates to the groin and testicle with retraction of this latter organ. Tenderness is elicited on pressure over the kidney. There is irritability of the bladder and diminished excretion of urine, which contains pus and possibly some blood. In the absence of urinary symptoms, abscess of the kidney, and particularly if it be a floating kidney, is necessarily more difficult to differentiate. In the latter instance, however, the tumor will be movable. I have recently operated on a case of acute suppuration of the kidney in which the urine was normal, and the diagnosis was made on the anatomic situation of the swelling. Nausea, sometimes with vomiting, is not an inconstant symptom.

6. *Perinephric abscess.* When the appendix holds a retro-cæcal position and an abscess forms, it may be mistaken for a perinephric abscess, but the absence of intestinal disturbance will be sufficient to clear the diagnosis.

7. *Nephritic colic.* To differentiate between nephritic colic and appendicitis is at times difficult, owing to the fact that in the latter condition there is in rare and exceptional cases pain referred to the umbilicus, retraction of the testicle, associated rectal and vesical tenesmus, and painful micturition. This error, however, could only occur in the early stages of appendicitis, as the symptoms of the later stages are so entirely dissimilar. I recall the case of a physician, in which the diagnosis of renal colic had been made, and in which the ureter was supposed to have been ruptured by the passage of a calculus. The autopsy revealed a gangrenous and perforated appendix with diffuse suppurative peritonitis.

8. *Hepatic and perihepatic abscess.* Appendicitis can only be confounded with abscess of or about the liver when, late in the disease, a circumscribed collection of pus is in close relation with this organ, and when the appendix is post-cæcal and points toward the liver. The previous history, the hectic temperature of hepatic or perihepatic abscess, and the pain referred to the shoulder will be sufficient to establish the diagnosis.

9. *Entero-colitis.* In this disease the colicky pain is more marked, and there is present a mucous diarrhœa and extreme collapse.

10. *Neuralgia of the right iliac fossa.* Neuralgia of the right iliac fossa is a condition associated with localized intermittent pain, which is relieved rather than increased by deep pressure. A case has been reported by Shrady of a medical man whose appendix was removed under these circumstances.

11. *Extra-uterine pregnancy.* From extra-uterine pregnancy the diagnosis is made by a history of pregnancy; and, along with the rupture of the sac and production of a pelvic hæmatocele, there is profound collapse followed by a discharge of blood and decidual shreds.

12. *Typhoid fever.* From typhoid fever appendicitis can be differentiated by the frequency of the pulse, the absence of the characteristic temperature, the coated tongue with red border, epistaxis and headache. The presence of spots does not necessarily indicate typhoid fever, as spots may be present in septic conditions.

13. *Hip-joint disease.* The presence of the characteristic deformity, inability to execute the normal movements of the joint, pain referred to the knee, arching of the lumbar spine when the limb is brought into the fully extended position, and absence of intestinal symptoms, should settle the diagnosis.

14. *Abscess of the abdominal wall.* Between abscess of the abdominal wall and that caused by appendicitis, there should be but little difficulty in arriving at a correct conclusion. If the collection is in the superficial fascia it will be circumscribed, while if between the abdominal muscles it is likely to

be diffused. The purely local character of the abdominal abscess, the swelling moving with the abdominal walls, the absence of intestinal symptoms, the presence of local and constitutional evidences of pus, coupled with the history of the case, should be enough to render a differential diagnosis possible.

Prognosis.—The prognosis of appendicitis depends not only upon the character of the attack and the complications which may supervene, but upon the character of the treatment instituted.

Our present knowledge gives early operation the first place in the treatment. Many cases of simple catarrhal appendicitis subside under careful and rational medical supervision; still, in the great majority, this primary attack only leaves the patient predisposed to another which is more severe and more dangerous than the original one. In my last series of cases, forty-five of which I have operated upon, forty-two had had previous attacks. In other cases which start up acutely there is over the diseased organ a gradual decrease in all the symptoms excepting tenderness, thus leaving the patient in a subacute condition. In this subacute condition the appendix may, and frequently does, go on to perforation and gangrene, without a corresponding increase in the symptoms. When the appendix has been removed a second attack is an impossibility, so that I believe that operation gives the patient the only chance for permanent recovery.

When the collection of pus is circumscribed, the general peritoneal cavity being walled off, the prognosis is generally favorable. If the general peritoneal cavity be early infected the prognosis is grave, although if operation be at once resorted to there is a possible chance of recovery; while if the infection be late or operation deferred, a fatal termination is inevitable. When a circumscribed appendiceal abscess ruptures and evacuates its contents into the bowel, recovery usually follows, while evacuation into the bladder is fatal in about 50 per cent. of the cases. The age of the patient is a decided factor in the prognosis, as the disease is more

fatal in the very young, on account of the lessened powers of resistance.

Complications and Sequelæ.—By far the most important and frequent complication of appendicitis is peritonitis. Inflammation or phlebitis of the right iliac vein, or thrombosis associated with œdema of the corresponding lower extremity, is a complication sometimes met with in the localized suppurative variety of appendicitis, and if a fragment of the thrombus be carried along the general circulation, this may result in foci of septic pneumonia or septicæmia.

If the appendix lies over the iliac bloodvessels, and undergoes ulcerative destruction, it may cause necrotic change in the walls of the bloodvessels, and result in fatal hemorrhage. Abscess of the liver, purulent collections in the pleura and pericardium, may also result from portal thrombosis or directly from the general circulation. Appendicitis may be a complication or an accompaniment of hernia. When a localized appendiceal abscess ruptures into the bowel or into the bladder, there is renewed infection from these avenues. Fecal fistula is a frequent sequel of appendicitis.

Treatment.—I have made no attempt to formulate a rule by which we are able to discriminate which class of cases will terminate as simple appendicitis, or go on to perforation or gangrene. There are some who claim to be able to foresee which cases of appendicitis will eventuate in recovery or assume the gangrenous type, or to recognize the onset of this dangerous complication. This is, I believe, an assumption which cannot be substantiated by experience. My experience is that the surgeon is often called to see cases in which the unfortunate patient, according to the attending physician's statement, has been doing well up to a few hours previously, only to find the evidences of general septic peritonitis, which invariably foretells the almost inevitable result. Is it to share the responsibility? Certainly not with the hope that operation promises recovery. Again, why should we trust to chance when we have positive means or relief in immediate operation, if the treatment to which I shall refer further on fails to accomplish

the desired end? Granting that the majority of the cases of simple appendicitis will temporarily recover under careful and well-directed medical treatment, are we justified in allowing the minority to perish when we have positive means at our disposal to prevent the fatal issue?

The physician who has had the opportunity to observe many cases of appendicitis cannot but have impressed upon his mind the lesson of cause and effect—the cause being the primary attack, the effect the death from a subsequent attack.

While some may claim that they have never seen a recurrence, there are many who will bear witness to the fact that they have frequently seen subsequent attacks. My friend, Dr. J. M. Da Costa, recently related to me the history of a case in which the patient had had forty-seven previous attacks; in the forty-eighth attack he asked the surgeon who was in attendance with him to operate. The operation not only terminated favorably, but demonstrated the presence of a collection of pus within the appendix which was on the verge of rupture.

When advising operation I have been met by the statement from the attending physicians that they have seen patients recover from several recurrent attacks, and presenting this as an argument against operation. The patient just referred to, who had forty-eight attacks, certainly demonstrates to the unprejudiced mind that one more attack would most likely have resulted in rupture and septic peritonitis.

While this paper is upon the subject of "Acute Appendicitis," I may be permitted to digress to make the statement that of the thirty cases of chronic appendicitis upon which I have operated successfully this winter, the lesions present in the majority of the appendices demonstrated most clearly that an acute attack would most probably have resulted disastrously.

If operation is justifiable in the minority of cases, which without operation are fatal, is it not equally justifiable in all cases, considering that the majority which recover under well-directed medical treatment suffer subsequently from recurrent attacks, and many perish in the last attack?

In my experience operation, performed under strict aseptic

precautions by a surgeon familiar with the anatomy and the technique of the abdominal cavity, and immediately following the establishment of the diagnosis, will give a mortality practically *nil*.

If the medical man had the same opportunity to observe the ravages of an appendiceal inflammation ante-mortem as has the surgeon, particularly one who has operated many times, he would certainly be convinced of the importance of early operation; hence, I can understand the position of the conservative physician.

I will ask in advance those who will take exception to early operation, if they have ever had the opportunity to observe the treatment I advocate. Anyone who has seen a gangrenous appendix removed before perforation, and before a localized abscess or a general septic peritonitis has developed, could not fail to sanction prompt interference.

The first indication to be fulfilled in a case of simple appendicitis, or even of a suspected case, is to obtain free action of the bowels. This is best accomplished by the administration of castor oil, followed by salines, preferably Rochelle salts, or, if the stomach is not tolerant to salines, small doses of calomel. Free evacuation of the bowels—not drastic purgation—serves the purpose of clearing the alimentary canal of all foreign and irritating substances, such as particles of undigested food, etc., and relieves the congested bloodvessels, thus modifying the degree of the subsequent inflammation. Again, this course fulfils a most important indication in having the bowels in the most favorable condition for operation in the event of its necessity. I cannot emphasize the importance of this part of the treatment too strongly, as upon it, I believe, largely depends the chance of the patient's recovery. Certainly in my experience, the bulk of patients I have seen recover without operation have been those in whom this was most thoroughly carried out, and in the cases operated upon the largest percentage of recoveries have been those in whom the bowels were not allowed to be confined. The pain in appendicitis, which is colicky in nature, is due first to the muscular con-

traction of the appendix and the bowels, and in the majority of instances is provoked by irritation within both; therefore, is not the emptying of the intestines of their irritating contents urgently called for? Is not the bowel, in other words, asking to be relieved? This being so, will not purgatives take the place of an anodyne, a hypodermatic of morphine, which is often and indiscreetly given? The best anodyne consists in repeated doses of salts or calomel, which do more than any preparation of opium can do, both relieving and removing the cause of the pain. Free purgation should be continued throughout the attack, sufficient to keep the contents of the bowels in a liquid state as long as the tenderness and uneasiness in the appendix exists.

Absolute rest in bed, with careful feeding, should be enforced. Turpentine in the form of hot stupes should be applied, and will relieve the distention and pain in addition to acting as counter-irritants. The use of leeches and blisters is advocated by many in preference to purgation, to accomplish the depletion of the circulation. This is not in accord with my experience. Blisters are particularly objectionable, in that they macerate the superficial layer of the integument, increase the patient's discomfort, do not accomplish the results afforded by salines or calomel judiciously administered, and, one of the strongest objections against their use, they make a septic field for operation. This latter is also true of leeches.

Opium should not be given; it usually masks the character of some of the most important symptoms. Absolute rest in bed, with careful feeding and mild purgatives, generally relieve these cases within thirty-six or forty-eight hours. In those in which the symptoms do not abate, and the disease seems to be progressive, the appendix should be removed. When opium has been administered and the progressive character of the case is doubtful, it is better to err upon the side of safety, and remove the appendix at once. The condition present is usually beyond the powers of Nature to remedy, while in the hands of a surgeon who pays strict attention to the aseptic details of the operation the latter entails little risk

to life—less than that which is involved in even a mild attack of appendicitis that remains stationary, with all its possibilities of lymphangitis, infection of the peritoneal cavity, retained muco-pus within the appendix, and rupture of the latter into an unprotected peritoneal cavity, or ulceration and perforation from inspissated fecal matter, imprisoned by constriction within the cavity of the appendix, or from a gangrenous condition alone.

When the disease has gone on to the stage of pus-formation, and we are positive that such a condition exists, usually shown by persistent pain and tenderness which is intense on deep pressure, no time should be lost in opening the abdominal cavity, evacuating the pus, and removing the appendix at the same time. The less medication these cases receive the better, and the use of salines is especially contra-indicated. The limiting wall of the abscess-cavity is delicate, and the increased peristalsis set up by the saline may be sufficient to rupture the abscess wall, with resultant leakage of its contents into the peritoneal cavity and subsequent peritonitis.

In all cases in which the disease is not of a mild type at the outset, as indicated by the symptoms, I believe that operation is the only treatment offering any positive means of relief, and that it should be instituted as soon as the diagnosis is established. Salines, and this is more true of opium, are especially contra-indicated in this class of cases, in which there is no certainty as to the condition of the appendix at its attachment to the cæcum.

Rectal injections are contra-indicated for the same reason. I have recently seen a case in which a gangrenous appendix was forced away from its attachment to the cæcum by a copious injection which was emptied into the abdominal cavity, followed in a few hours by a septic peritonitis. Prior to the injection the patient's abdomen was flat, the temperature normal, and the pulse good.

Great care should be exercised when opening these abscess-cavities or infected areas that the general peritoneal cavity be not infected. The manipulation should be very gentle, as

a communication may readily be established. With a good assistant, who with gauze compresses is able to keep the field of operation well isolated from the rest of the peritoneal cavity, and also to keep the small intestines out of the way, the operation at this period of localized peritonitis, with or without sero-purulent collections, is both safe and advisable. It is almost impossible to avoid breaking away some of the adhesions, but with care the appendix can be removed without infecting the peritoneal cavity. After removal the general peritoneal cavity can be sealed off by properly packing with iodoform gauze previously sterilized.

When the appendix has been separated from the cæcum at its basal attachment by a gangrenous inflammation, if the opening in the cæcum is not too large and the surrounding area not devitalized, the margins of the opening should be cut away and the opening closed by the Lembert suture. On the other hand, if the opening be large, and the adjacent area is involved in this gangrenous process, it is better to wall off this portion of the cæcum, insert a glass drainage-tube into the abscess-cavity, and pack iodoform gauze around it, thus sealing it off from the peritoneal cavity. A fecal fistula will almost always follow, but this usually closes spontaneously. I have known cases, after the abscess-cavity has simply been opened and drained, to recover with a fecal fistula, the fistulous tract communicating with the tip end of a patulous appendix. This demonstrates the importance of removing the appendix when the abscess-cavity is opened.

When the abscess-cavity is post-colic, the incision should be made through the outer layer of the ascending mesocolon, the appendix removed, and aseptic drainage established through the loin. In a post-cæcal appendiceal abscess, especially if the appendix be the seat of gangrene, there is great danger of infection of the liver through the venules of the portal system which are in relation with the main layer of the ascending mesocolon. When the appendix runs over the brim of the pelvis and extends into the pelvic cavity, it is always better to drain with a glass drainage-tube, placed in the most dependent

portion of the pelvis. In case a secondary abscess should form and occupy the recto-vesical space in the male, it is better to drain it through the rectum, using a larger trocar and canula to penetrate it, and then insert an English catheter through the canula, while if it occupy the recto-uterine space, it should be emptied through the vagina. I think it is better to resort to this procedure than to reopen the original wound or open through the linea alba, on account of exposing the peritoneum to a second infection.

I have not been able to see the advantage of the linea alba as an avenue through which to remove the appendix, as advised by some operators.

From the fact that the appendix in its normal position lies in the right iliac fossa, the most direct route to it is through the right semilunar line or immediately to either side of it. By carrying the incision either through the rectus or the internal oblique and transversalis muscles, there is no doubt that the wound can be closed with greater security against the occurrence of a hernia, whether a series of buried sutures be used or not.

There is no question but that the Trendelenburg position offers advantages in certain cases, and that the evacuation of pus or drainage of an abscess, and the subsequent irrigation, are favored by turning the patient over on the affected side.

It has been my intention not to cite any of the numerous cases I have records of, knowing how tiresome this is to an audience, but while writing upon the treatment of this affection I was called to see a case which so clearly supports the position I hold relative to early operation, that I will beg of the Fellows the privilege of reporting it:

Friday night, April 27, 1894, Dr. Prendergast was summoned to see Mrs. O., thirty-five years of age, who on account of acute abdominal pain, accompanied by nausea, general abdominal tenderness, and constipation, was suddenly taken to her bed. The pain was so severe as, in Dr. Prendergast's opinion, to call for a hypodermatic of morphine. Upon careful inquiry a history of one similar attack, much less severe in character and occurring four months ago, was obtained. On the following day, Saturday, the bowels having been moved by purgatives, the patient thought herself to be more comfortable.

On Sunday the pain was more severe and was referred to the right iliac fossa; there was pronounced general abdominal tenderness with decided tympanites; the pulse was 100; the temperature 102°. The patient was referred to me for admission to the German Hospital. Upon being given a statement of the case over the telephone, I requested the patient to be prepared for operation. The examination of the pelvis was negative. Examination of the abdomen showed very evident general peritonitis and excessive tenderness to pressure over the site of the appendix. Operation demonstrated a small peri-appendicular abscess—an appendix which held a southwest position, turned upward and outward upon itself, gangrenous at its distal extremity to the extent of an inch, and adherent to the floor of the right iliac fossa by its tip. The mesentery of the appendix was so infiltrated and thickened as to resemble a second appendix. The general peritoneal cavity was not shut off. The pus was evacuated, followed by irrigation and removal of the appendix. The morning after the operation the temperature was 98° and the pulse 84. The patient was passing wind and the abdominal distention was rapidly disappearing. On Tuesday morning the improvement continued. On Wednesday the pulse was 68, the temperature normal, and the bowels moved the previous night. The abdomen was flat and improvement uninterrupted.

DISCUSSION.

Dr. J. M. Da Costa: I rise with some diffidence to speak on a subject of this kind; it is so largely surgical, and so many distinguished surgeons are present, that I am aware that I am addressing an audience of experts. But though the condition is undoubtedly in the main a surgical one, before the case gets into the hands of the surgeon the physician sees it, and to him as well as to the surgeon belongs the responsibility of declining operation or of urging it. I shall not take up time by going into the discussion of the whole subject, but merely speak of some matters that experience has made clear to me—an experience shared in many instances with noted surgeons, but of which I may summarize the results from a general and purely medical point of view.

In the first place, there is the question of diagnosis, which, after all, belongs more to the physician, as he sees the case early, and he has to decide whether or not it is one of appendicitis. I want to say distinctly that with what Dr. Deaver has so clearly stated of the valuelessness of the thermometer in the recognition of gangrenous or purulent appendicitis, I am in accord. I could cite examples of gangrenous appendicitis, with pus around the appendix, in which the temperature was almost normal. In one instance which I saw with Dr. Morton, gangrene of the whole appendix was found, but at no time did the temperature go above 99.1°. I should also like to

say that while the thermometer does not help us, the pulse does not do so much more, and that, after all, it is by the whole history, and particularly by the kind of pain, that we make up our minds as to what is the matter; I was almost going to say by the locality of the pain, but this is true only up to a certain point. In one of the most striking cases with which I was ever connected the pain was over the liver. It is sometimes epigastric; in the case that I saw with Dr. Deaver the pain was on the left side, and most puzzling it was to determine this to be an instance of appendicitis. Looking at the admirable paper of Dr. Fitz, I find that pain in the left iliac fossa occurs in only 1 per cent. of the cases. There is one seat of pain rarer, namely, pain in the hip and groin, which is so rare that it happens in only $\frac{1}{2}$ of 1 per cent. The seat of pain, therefore, must not mislead us. It need not be in the right iliac fossa.

Another question in diagnosis is as to the presence of pus. If we only had a sure test indicating the presence of pus in appendicitis, it would be a most valuable addition to the whole subject; but we have not. I have already said that the thermometer does not help us. The sense of resistance and the fulness are of some value; so is severity of recurring pain. But the great tenderness that exists in cases in which pus is present is, perhaps, the most certain sign; yet it is not an absolute sign. In rectal examination with the view of detecting a swelling, I have been disappointed except when there is a considerable collection of purulent material. If I were asked what single sign is there of pus in appendicitis, I should say that the only thing, barring the marked tenderness, that comes near being a distinctive sign is the great variability of the symptoms. You will find the pulse up, then down; the respiration embarrassed at one visit, less so at another; there will be peculiar fleeting eruptions on the skin; there may be coarse capillary injection here and there. These signs, which show a septic condition, are probably the most certain, although, after all, not very certain, signs of purulent collection.

Looking to some questions of special diagnosis, typhoid fever is of a great deal of interest. One of the most remarkable instances that I ever saw was one Dr. Morton and I had together, in which there had been a previous attack of appendicitis, Dr. Morton having attended the man through it. The pain was localized in the right iliac fossa, and strongly pointed to a recurrence of the previous malady, but the temperature was 104°, and in both of our experiences this was so unusual in appendicitis that the case was allowed to go on from day to day without surgical interference, and at the end of the sixth day lenticular spots appeared on the abdomen, the bowels became loose, and the case ran the course of typhoid fever. Here the height of temperature was so great that, notwithstanding a clear knowledge of the history, Dr. Morton most judiciously held his hand. Of course this difficulty will not arise when we have the temperature-chart and can trace the characteristic rise of typhoid fever. But we are often not called in at the

onset, and we have to judge of the case as it stands. I would state that early height of temperature, a temperature remaining above 102°, would be an important point against appendicitis and in favor of typhoid fever. On the other hand, severe and recurring pain in the right iliac fossa is against this. With regard to the spots which have been stated to exist in appendicitis, and which are supposed to indicate pus, these do not have bearing on the question of differential diagnosis. They are not the characteristic eruption. They are not rose-colored spots of definite life; they are coarser, irregular, and very fleeting.

The question of diagnosis with reference to obstruction of the bowels, as I know by experience, is sometimes very difficult; it seems almost impossible to solve the question. Every single symptom, including scanty urination, that exists in appendicitis may be present. Generally, however, the absence of fever early in the case, the markedly distended and tympanitic abdomen, the obstinate constipation, the diffuse rather than localized pain, its lesser violence, distinguish the intestinal obstruction.

I turn from these points of diagnosis to some points of treatment. It would be out of place here to go into a full discussion of the medical treatment of acute appendicitis. I will only state that in the main I agree with the doctrine that free purgation is advantageous, and that the use of opium should be very moderate and ought to be avoided, certainly early in the case, if the pain will permit us to do so. There is a remedy which, in the early stages, I have known to be of advantage—the application of ice over the region of the appendix. If time permitted, I could give the detailed history of several cases in which the symptoms of acute appendicitis disappeared under this treatment. However, I pass on to the important point where surgery and medicine touch—the question of operation. While we have to defer most largely to the judgment, to the skill, and to the acquired knowledge of the surgeon, nevertheless the physician for his part also comes to certain definite conclusions. I cannot go as far as Dr. Deaver in thinking that every case of appendicitis had better be operated on. It seems to me that if we take this view, a person with appendicitis will go to bed not with the proverbial sword of Damocles suspended over his head, but with the scalpel of the surgeon already on his abdominal wall. I believe that the majority of cases of appendicitis in the first attack recover under medical treatment. This is certainly true when no perforation has happened. I am quite willing to think that what we here speak of to-night may be antiquated in six months. The wonderful technique, the ever-growing skill of the surgeon, and the fact that things become possible to-morrow which are not possible to-day, may make us all before long come to the conclusion that the percentage of risk is much less in operating in many instances than in trusting purely to medical treatment. I am free to admit this; but, speaking from our present knowledge, I think that it is right to select only certain cases for operation. I give the results of my personal experience, much of

it gained in association with surgeons. In the first place, I believe that, in addition to perforative appendicitis, it is our duty to operate early on every case of appendicitis in which there are signs of abscess, or in which there is good reason to think that pus exists, no matter how little. Let us rather occasionally be mistaken than run the risk of not operating. Therefore, if we could come to greater accuracy in the diagnosis of pus, I hold that the main point in regard to operation would be solved.

I also think that while we may and ought to have much hesitation as to the propriety of operating when appendicitis occurs for the first time, yet with every subsequent attack this hesitation should vanish. The risk becomes greater and greater. I think that when a person has had more than one attack, certainly when he has had several, especially at short intervals, it would be quite right to consider an operation as his best chance. I say this, for I have seen the results of delay. It is true the character of the attacks will have much to do with the issue. If the appendicitis be merely catarrhal, or the result of something ingested that is readily gotten rid of, a number of attacks may be borne with impunity. But the parts become altered and weakened by the repeated disorder, and, unfortunately, diagnosis has not reached the point that we can be sure of the tissue-changes that have happened and gauge the power to withstand ulceration.

Again, when there have been signs of disturbance of a chronic kind in the lower part of the abdomen, although there has been no distinct history of appendicitis, I think that it is better to operate if a well-marked acute attack comes on. I recall a case in the Pennsylvania Hospital in which a young man, with obscure abdominal symptoms and some thickening in the right iliac fossa, was attacked with acute appendicitis. He was doing so well that I could not make up my mind to advise operation, but perforation and rapid peritonitis followed. It was afterward learned that there had been a previous appendicitis, and the autopsy showed that an early operation would most probably have saved him. I saw a similar instance in a boy eleven years of age.

There are some points that arise with reference to the time of operation in acute appendicitis which are purely matters for the surgeon. But that we ought to operate if the case goes beyond a certain day, an opinion often promulgated, I cannot agree to. I have seen cases that have gone on for a long time, for eleven days, for fourteen days, and yet perfect recovery has taken place. Therefore I do not think that the absolute law which is laid down by some, that you must operate on or soon after the third day, is a law that holds good. We must judge by the character of the case, the general symptoms, the way it is progressing, the liklihood of the presence of pus, rather than by the mere number of days.

In conclusion, I will state that I know of no question in practical medicine that is a more important, a more solemn question than this one of operation in appendicitis. On the one hand, you feel that you may be led into an

operation that is unnecessary, and that in the best hands is attended with some risk, and, on the other hand, you know that delay may be fatal. It is, indeed, a trying question, and we must solve it according to our best judgment in individual cases with close thought, putting the operation off if we safely can, but remembering the issues at stake. If we err let it be on the side of early operation rather than on that of too long delayed operation.

Dr. George E. Shoemaker: There is no symptom which is constant. Dr. Deaver referred to tenderness as always present, but Dr. Da Costa has spoken of the uncertainty of this point. I wish simply to mention a case which passed out of my hands into those of one of the best surgeons in the city, who could not find either rectal or abdominal tenderness. No operation was done, and the patient died with a gangrenous appendix. Exceptionally there may be no local peritonitis, and rapid gangrene abolishes sensation in the affected part.

Dr. G. G. Davis: Referring to Dr. Da Costa's allusion to typhoid fever, I will mention a case that was brought into the German Hospital. The patient was a Hungarian from whom no particulars could be obtained, and evidently in a very critical condition. There was some fever, a tympanitic abdomen, and dulness in the right iliac fossa, and he was suspected to have appendicitis. The abdomen being opened a plastic, purulent peritonitis was found in the neighborhood of the cæcum. The appendix was found only slightly inflamed, and as the condition of the patient did not warrant further search for the cause of the peritonitis, the abdomen was drained and closed. The patient died on the following day, and post-mortem examination showed that it was a case of typhoid fever with two perforations, one four inches and the other ten inches from the ileo-cæcal valve.

Dr. D. D. Stewart: For several years past I have been interested in the subject of the diagnostic value of peptonuria, and more especially as it applies to the recognition of appendiceal abscess. As is well known in all cases in which pus is present in any quantity in the system in a situation in which disintegration and absorption of the products of disintegration of the leucocytic cellular elements are occurring, this so-called peptonuria is likely to exist. Unfortunately it appears that so-called peptonuria is not limited to pyuric conditions, and apart from its occurring with abscess, and in other conditions characterized by the abundant presence of pyuric elements, as in the resolution stage of pneumonia, in articular rheumatism, in phthisis, and in purulent meningitis, it may be also present in certain conditions of deranged digestion. The well-known researches of Wright especially have shown that the body called peptone in the past is one of the albumoses and not peptone at all, and that in reality it is derived from the disintegration of the nucleo-albumin of the leucocyte or pus cell. The examination for this so-called peptone in the urine, as a product of the disintegration of leucocytes away from the urinary passages, is beset with difficulties. It is almost never present in quantity to admit of its detection by

the biuret test without great concentration of the urine. Hoffmeister's method is the only very accurate one for its detection, and its application consumes much time. Of course all other albuminous bodies likely to be present have to be considered and excluded. I had begun an extended investigation into the subject of peptonuria several years ago, hoping to arrive at some definite opinion as to its diagnostic value in conditions of latent suppuration, but I had to abandon a general research after a time, as I could not single and alone carry out the enormous amount of work the investigation demanded. I was and still am especially interested in the question of recognition of appendiceal abscess through the examination of the urine for peptone. The Fellows may recall my asking for specimens from suspects, through the *Medical News*, some two or three years ago. I should have been very glad to continue to pursue this limited branch of the subject, but I could not succeed in interesting anyone save Dr. Keen in the question among those meeting many cases in which latent appendiceal suppuration was in question.

Dr. Thomas S. K. Morton: I have here an appendix which I removed last Sunday, and it shows the danger which lurks in some of these cases. The patient, a man aged twenty-seven years, had a mild first attack of appendicitis five months ago. On Wednesday last another attack of the same nature began with acute pain in the right iliac region, in a few hours extending over the abdomen, with great distention, uncontrollable vomiting, and moderate temperature. When I saw him in consultation on Sunday, he was in a condition of collapse, and I told the family that he would probably die on the table if operated upon. However, they insisted that the only chance we had to offer should be taken, and I opened the abdomen and fully three pints of putrid pus escaped. This was thoroughly washed out and a glass drainage-tube inserted. The condition was general purulent peritonitis. Oxygen was administered and he reacted very well, but during the night suddenly collapsed and died, I presume from toxines absorbed from the pus. The great interest in the case is the character of the perforation, which is a very small one immediately at the point of junction of the appendix with the colon. No foreign body was present.

Dr. Judson Daland: I was interested in the description of the appendix preceding perforation. It occurred to me that perhaps sufficient stress was not laid upon the condition of acute catarrhal appendicitis and the method by which it may produce ulceration and perforation of the appendix. When the inflammatory swelling of the proximal end of the appendix is sufficient to completely close it, the interior of this closed tube is filled with *retained* catarrhal discharges which in time become more irritating producing ulceration and later peritonitis with or without perforation. As to the presence of foreign bodies in the appendix, which all have seen in autopsies where no inflammatory trouble is present, I think these act in the same way: the cæcal opening of the appendix being closed, there occurs catarrhal inflam-

mation, ulceration, and finally perforation, but as long as it is patulous no symptoms occur.

I also think that the diagnosis of appendicitis is not always easy, but on the contrary at times very difficult or impossible; I recall a number of cases illustrative of this statement. In one case, a gentleman apparently in good health with slight rise of temperature to 100°, complained of colicky, abdominal pains not localized, three acts of vomiting, and went into collapse, and died in less than three days. The autopsy showed gangrenous appendicitis with perforation.

In New York so much has been said regarding this subject that the danger is rather in the direction of diagnosing its presence when absent rather than the contrary. This was illustrated by one of my patients who has had repeated attacks of biliary colic; while in New York a slight attack occurred, and the diagnosis of appendicitis was at once made.

With regard to operation on all cases of appendicitis, it seems to me that there is little doubt that almost all cases must be operated on so soon as diagnosed. Furthermore, in many abdominal conditions where the diagnosis cannot be made and the symptoms are grave, an exploratory laparotomy should be performed without delay.

Dr. John B. Deaver: In conclusion, I wish merely to say that Dr. Da Costa slightly misunderstood my remarks. I do not advocate operation in every case of appendicitis, but only in those which do not yield to the treatment described.

ON THE REACTIONS OF NUCLEO-ALBUMIN (ERRONEOUSLY STYLED *MUCIN*) WITH THE COMMONLY EMPLOYED URINARY ALBUMIN TESTS; THE DIFFICULTY OF DISTINGUISHING THESE REACTIONS FROM THOSE OF SERUM-ALBUMIN, GLOBULIN, ETC.

Remarks as to the Occurrence of a Normal, Constant Albumin-trace in the Urine.

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[Read May 2, 1894.]

In a paper[1] read before the Society for Clinical Research, I drew casual attention to certain fallacies attending the use of delicate tests for the detection of albumin in urine, laying stress on one especial source of error which I regarded as the most important and misleading. This related to the frequent presence normally of nucleo-albumin in amount sufficient to react to nearly all of these tests, such as Tanret's, Millard's, and Sebelein's reagents, and to all other similar test solutions containing tannin, mercury, or a vegetable acid. This also applied to picric acid, especially the citrated solution, to metaphosphoric acid, but particularly to trichloracetic acid, now much in vogue as a reagent for the detection of small amounts of serum-albumin in the urine.

[1] "A Serious Fallacy attending the Employment of Certain Delicate Tests for the Detection of Serum-albumin in the Urine, especially the Trichloracetic Acid Test," Medical News, May 5, 1894.

I pointed out that a reaction could so commonly be obtained from several of these, with the urine of the healthy, that unless this were regarded, it was unquestionably better in clinical work to depend upon the less misleading, if also less delicate time-honored methods, especially that by ebullition with, if necessary, the addition of acetic acid.

I had at one time regarded the reaction so frequently obtained with healthy urine, as indicative of the existence of a physiological albuminuria, but within the past year or two the result of certain experiments of my own and the work of others in the same field has rather tended to convince me that in the vast number of cases the reacting substance was a mucoid body probably originating from the cellular elements of the extra-renal passages. My reason for so believing I have entered into elsewhere,[1] so need not here detail them. Lastly, re-experimenting with the delicate tests, in investigating certain cases of non-albuminuric nephritis, I have been especially interested to determine if the nature of the reaction often obtained by several of the very delicate tests, and presumably not due to serum-albumin or to globulin, could be exactly determined, and whether it be a mucoid body settled.

Exact experiments with various percentages of artificially added nucleo-albumin to albumin-free urine seemed the most satisfactory mode of ascertaining with certainty the nature of its reactions, and their differences from those of serum-albumin, serum-globulin, the albumoses and peptone, bodies with which there is no doubt that nucleo-albumin has repeatedly been confused by many of the most accurate and perhaps all of the careless workers in urinalysis. As stated in the paper previously referred to, urinary nucleo-albumin, which is now regarded as identical with the nucleo-albumin of the bile, is not true mucin, though still improperly so termed by many who are unaware of the important differences in these bodies which separate them both chemically and physically. Briefly, these bodies are distinguished, in that concentrated solutions of

[1] American Journal of the Medical Sciences. Medical News, April 14, 1894. Ibid., May 5, 1894.

mucin are mucilaginous and thready; with acetic acid mucin gives a precipitate insoluble in excess, and that mucin, as a glycoproteid substance, after prolonged ebullition with dilute mineral acids yields, besides an albuminous body, a carbohydrate which reduces copper hydroxide.[1] Magnesium sulphate, also, is without precipitating effect with solutions of mucin, totally unlike the case with solutions of nucleo-albumin. These differences in reaction have enabled it to be shown that it is doubtful if a true mucin is ever present in the urine, and that the reacting body is in reality nucleo-albumin. This fact is of the highest clinical importance, since, apart from a local nucleo-albuminuria,[2] all the indications point to the fact that a nucleo-albuminuria, the nucleo-albumin of which is underived from any portion of the urinary passages, is of no uncommon occurrence. The clinical aspect of nucleo-albuminuria cannot now be discussed; suffice it here to state that it is a subject worthy of attention, the study of which is in the line of the elucidation of not a little that is obscure concerning the question of albuminuria without nephritis.

In the course of recent experiments with urine from the healthy, specimens of day and food urine were often encountered which, with proper precautions to exclude precipitation of urates, gave a sharp cloud after appropriate treatment with acetic acid; but it was always found impossible to obtain by filtration, although dilution was first practised,[3] sufficient of a precipitate in this manner to be utilized for experiment, such really small amounts did the precipitate represent.

As urinary nucleo-albumin is unobtainable in trade, the experiments were made with nucleo-albumin (Merck's) de-

[1] See Hammarsten: Treatise on Physiological Chemistry. Sheridan Lea: The Chemical Basis of the Human Body. Neubauer and Vogel: Analyse des Harns. Gamgee: Physiological Chemistry of the Animal Body.

[2] That is, nucleo-albumin derived from the cells of some part of the urinary tract.

[3] Because of the soluble effects of certain of the urinary salts, it is more easy to remove the mucoid precipitate produced by acetic acid in dilute urine than in one of ordinary gravity. It is also on this principle that the interesting fact pointed out by Reisser is explained, that if to a specimen of urine which may be clouded by the addition of acetic acid, a second is added which is not so affected, no cloud may be produced by testing the mixture.

rived from bile. This substance is regarded as identical, both chemically and in point of its various reactions, with nucleo-albumin occurring in urine. This body was found to be very slightly soluble in distilled water, and much more soluble in neutral or in alkaline saline solutions. With none of these in concentrated solution did it have a mucinous appearance, and the other tests, including ebullition with a dilute mineral acid, and subsequent testing the precipitate for a reducing substance, showed that the body contained no true mucin. In the solid state it gave the xanthoproteic reaction, but reacted doubtfully to Adamkiewicz's reagent, and gave no response to Millon's, Liebermann's, or to the biuret test. This last was also examined for in strong solution, without result. This lack of reaction to certain of these color reagents is interesting not only as a point in separating the nucleo-albuminous response from that due to other albuminous bodies, but also in indicating that the nucleo-albumin employed in the experiments made by Müller, who obtained all the color reactions, was contaminated with a second albuminous body.

Acting upon the fact that neutral salts, such as NaCl, AmCl, Na_2SO_4, and sodium acetate, serve to maintain the nucleo-albumin of the urine in liquescence, no difficulty was encountered in obtaining a perfectly clear solution of this substance. The diluents used for the experiments were saline solutions of distilled water and normal urine; the former was prepared to contain approximately the amount of mineral salts represented by NaCl, and a less amount of sodium acetate, that exist in urine of ordinary gravity. No allowance was made for urea, as this is regarded as being without auxiliary solvent effect. Specimens of very clear filtered morning urine from normal individuals were employed which, unfiltered, gave no reaction with trichloracetic acid until after five minutes' contact. Nucleo-albumin was added in proportions varying from approxi-

[1] See Neubauer and Vogel (loc. cit.). It is there stated that Müller obtained all the color reactions with nucleo-albumin. The varying results there given can only be so explained. It is stated that one observer noted that heat was without effect upon a nucleo-albumin solution, and a second the contrary. The experiments I detail were made in a manner precluding error of this sort.

mately 0.4 per cent. to 0.02 per cent. In the preparation of the solutions, this substance was first triturated with a small quantity of the diluent, subsequently thoroughly agitated with the total amount, and on each occasion thoroughly boiled for some minutes.[1] After a large number of filtrations, a clear solution could be obtained. When an amount upward of 0.4 per cent. was added, some slight residue always remained in the filter; with smaller portions, solution of the whole quantity was readily obtained, although filtration, in removing turbidity naturally coincidently withdrew a certain amount in fine suspension.

A very large number of experiments, approaching several hundred, were made with these solutions. A variety of tests in common use for the detection of albumin in urine were employed: ebullition with and without the addition of acetic acid; nitric acid; acetic acid and potassium ferrocyanide; metaphosphoric acid; plain and acidulated saturated solution of picric acid; Millard's reagent; trichloracetic acid, and a test solution of mercuric chloride with tartaric acid, a newly introduced reagent regarded of greater delicacy than Tanret's solution. Besides the employment of the color reagents, notably the biuret test, the behavior of magnesium and ammonium sulphate in neutral solution was also ascertained.

The reactions with acetic and citric acid should be first mentioned. Citric acid was employed in both saturated and in dilute solution. With these in all the various strengths of nucleo-albumin solution experimented with, a sharp contact ring was obtained with more or less diffused haze above. Acetic acid was used in the form of the liquid glacial acid, and in 2 per cent. and also in 25 per cent. strengths. The reaction with acetic acid was similar to those obtained with urines containing the mucoid body. The acid was applied by the contact method, and by the addition of small amounts to

[1] Lest ebullition have any decomposing effect upon the nucleo-albumin, altering its character, which, however, was not anticipated, in the form of menstruum employed, all the experiments detailed with the boiled solutions were repeated with solutions of similar strength prepared without heat. The results were identical. This likewise showed the absence of contamination by ordinary albumin, more than traces of which would, of course, be removable by boiling.

10 c.c. or more of the test urine, a contact ring was obtained with all strengths of acid, with cloud above; though a less marked contact reaction occurred with the undilute glacial acid, in which latter, indeed, nucleo-albumin can itself be dissolved. A cloud occurred instantly on the addition of small amounts of acid to the body of a few centimetres of test solution, its intensity and rate of appearance varying directly with the amount of nucleo-albumin present, and inversely to the proportion of salts.

A reaction occurred to HNO_3, employed by the contact method with all strengths of nucleo-albumin solutions. It was still apparent as a slight contact cloud to a dilution giving but trifling haze with plain picric acid. With solutions approximately 0.1 per cent. or more, though carefully underlaid by the acid with a capillary pipette, the whole stratum of solution soon became opaque, and, after standing a short time, a deeper, opaque ring was found at the point of contact of the two liquids. With less strengths than 0.1 per cent., such as 0.08 to 0.05, it was easy to obtain an instantaneous contact reaction quite like the ordinary serum-albumin ring, although it lacked the appearance of denseness; the latter, if of any depth, usually has little or no opacity above, with these strengths of solution so tested.

A slight contact cloud was still apparent with the weakest solution used, approximating 0.02 per cent., a dilution which gave but a faint contact haze with unacidulated picric acid after standing a few moments. A few drops of fuming HNO_3 to a few centimetres of urine also produced a slight opacity in urine containing this same dilution of nucleo-albumin.

A combination of acetic acid and potassium ferrocyanide, a test for serum-albumin in the urine first proposed by Pavy, and now much in use, gave the ordinary albumin reaction, entirely indistinguishable from that of serum-albumin.[1] This

[1] Even so careful a chemist as Hammarsten (Text-book of Physiological Chemistry) does not speak of the similarity of reaction of nucleo-albumin and serum-albumin to either metaphosphoric acid or to the acetic acid and potash ferrocyanide reagent, in recommending these latter for the detection of albumin. No writer refers to confusion arising by the employment of metaphosphoric acid.

reaction was still apparent in the least strength tested. The reaction was decided at 0.05 per cent., and was still apparent at 0.025 per cent.

The various strengths of nucleo-albumin solutions also reacted similarly to metaphosphoric (glacial) acid. This reaction was in no way distinguishable from that of serum-albumin.

With considerable amounts of nucleo-albumin present, the whole solution above the piece of glacial acid became almost instantly opaque. With small amounts an almost instant contact opacity occurred, typically such as is produced by serum-albumin, and gradually diffused from the portion of acid throughout the fluid. If but traces of nucleo-albumin were present, but still sufficient to be apparent to acetic acid, though scarcely appreciable to unacidulated picric acid, the contact response was delayed, not appearing for from two to three minutes, but soon becoming decided with longer contact.

A decided reaction was obtained with saturated solution of picric acid, and with the citrated solution, indistinguishable in appearance from the contact response with ordinary serum-albuminous urine. The ring was most sharply defined when the nucleo-albumin solution was of not too low a gravity (above 1010). No reaction save a tardily appearing contact haze occurred, however, to unacidulated picric acid with a nucleo-albuminous dilution approximating 0.02 per cent., although the citrated picric acid still gave a ring, and response was still evident to metaphosphoric acid, and very markedly to trichloracetic acid, to Millard's, and to the mercuric test.

I had before shown that unacidulated picric acid solution reacted to the urinary mucoid body,[1] which is contrary to the view of Sir George Johnson. These experiments finally settle the question.

With Millard's reagent nucleo-albumin solution continued to give a sharp contact reaction with a dilution at which plain picric acid had entirely ceased to react.

With trichloracetic acid up to the weakest dilution of nucleo-

[1] By the removal of the reacting body with acetic acid in the cold. See American Journal of the Medical Sciences, December, 1893; Medical News, April 14, 1894.

albumin employed, a sharp contact reaction was obtained. With a dilution approximating 0.025 to 0.04, with which unacidulated picric acid gave no more than a contact haze, a markedly broad, opaque ring was apparent with trichloracetic acid.

With the tartrated mercuric reagent similar reaction was obtained as that of the others, although it was found that this reagent did not compare in delicacy with trichloracetic acid.

It is now of importance to study in greater detail the reaction of nucleo-albumin with these albumin tests, and to determine, if possible, how they may be differentiated from the reaction of serum-albumin, serum-globulin, and from the albumoses, but especially from the first named. It will be recalled that as thorough ebullition was invariably practised with the acid artificial or natural urine in preparing the nucleo-albuminous solutions, more than traces of ordinary albumin could scarcely be present; and that as it was necessary to repeatedly filter to obtain a clear solution for testing, more than traces of the albumoses, or so-called urinary peptones, if present, would also be removed, since filtration of the solution was repeatedly done not only while hot but after it was thoroughly cooled.

The likeness of nucleo-albumin to urinary albumose or peptone is striking in several particulars. That in the past it has been often mistaken for it, I have no doubt, in view of the identity in many of the reactions of the two. This curious similarity in reaction of these bodies probably depends upon a close kinship. As Wright[1] and others have shown, nucleo-albumin is identical with cell-fibrinogen, which under the influence of ferments (proteolytic) is decomposed into a nuclein moiety and albumose, or so-called peptone, the last of which is not infrequently present in the urine in cases of active leucolysis. It is conceivable and probable that traces of the albumoses are also present in the urine in conditions leading to proteolysis in the bladder of the urinary nucleo-albumin,

[1] See a paper on "Tissue- or Cell-fibrinogen in its Relations to the Pathology of the Blood." A. E. Wright, Lancet, March 5, 1892.

through the action of pepsin and trypsin, which are normally present in the urine, and by the aid as well of formed enzymes. But that nucleo-albumin is not identical with the albumoses or peptone is shown by its ready precipitation by acetic and other of the vegetable acids, and by the fact, as I have ascertained, that it will not give the biuret reaction. This last seems a very positive mode of differentiating it from the other albuminous bodies.[1]

Its differentiation from ordinary peptone is quite easy, as I have found that by saturating hot urine containing nucleo-albumin with ammonium sulphate all traces of nucleo-albumin are removed, so that no response can be subsequently obtained with trichloracetic acid.

Its similarity in several points to serum-globulin is also quite striking. Its presence in the urine, I believe, has often been mistaken by those who are unaware that globulin very rarely, if ever, exists without the coincident presence of a greater amount of serum-albumin.[2] Like serum-globulin, if mucoid nucleo-albumin is present in some amount in the urine, as I have found is common in specimens of the day urine (after food and exercise), it may not infrequently be precipitated from solution by considerable dilution of the urine with water, lessening the soluble effect of the urinary salts, or by dialysis to a very low specific gravity; or, like globulin, it may be readily thrown down by magnesium sulphate after neutralization with ammonia, and separation of the earthy phosphates.

A strikingly interesting and important feature of the reactions of mucoid nucleo-albumin to the albumin tests is the influence of heat. Although the presence of this body may be readily shown by the intelligent use of acetic acid or other of the vegetable acids, in the cold, yet without the assistance of heat it would be quite impossible by any test, were the re-

[1] It would become a clinical mode of differentiation of great value were the biuret reaction for albumin one of any delicacy.

[2] This seems well established, and yet not infrequently in urine reports of cases it is noted that serum-globulin but no serum-albumin is present. In these instances the reacting body is probably nucleo-albumin, which, I believe, is nowhere noted as likely to be a confusing factor.

action decided, save, perhaps the biuret test, to definitely assert that serum-albumin is not also present in some amount.[1] Heat with every test employed save two—the metaphosphoric acid, and acetic acid and potassium ferrocyanide—tends to diminish a nucleo-albumin reaction if the amount of this body present is in moderate or small percentage, such as is likely to be encountered in the urine. This result was invariable with a percentage upward of 0.15. With the presence of greater amounts, the soluble effects of heat were much less apparent; and with a percentage of 0.3 to 0.4, heat was either without influence or slightly increased the reaction previously obtained. This effect, also, I have found varies in degree with different specimens of the diluent of approximately the same degree of acidity containing not only different strengths of nucleo-albumin, but the same amount. The cause of this variation seems largely due to the character and amount of salts present.[2] Albumin-free[3] urines, and artificial urines experimented with, showed this variation with different percentages of nucleo-albumin. The reagents, whether HNO_3, picric acid, trichloracetic acid, or others, seem to hold, after precipitation, a portion of the nucleo-albumin, so that a temperature approaching the boiling-point, even with very small percentages of nucleo-albumin present, will not entirely dissipate the precipitate.

Heat was most commonly applied in the manner detailed in the former paper,[4] by immersing the test-tube in hot water to a point beyond the upper line of fluid. In the trichloracetic acid experiments there mentioned the bath was not always quite at 212° F., as so high a heat was not thought necessary.

[1] This is important, in view of the frequent coexistence of serum-albuminuria with a so-called mucinuria (nucleo-albuminuria).

[2] It is interesting to note that with approximately a 0.2 per cent. nucleo-albumin solution, containing NaCl, and a less amount of sodium acetate of a sp. gr. of 1017, the boiling-point has a slight precipitating effect. A further addition of a small amount of sodium acetate entirely prevented this precipitation in a second portion. Even without the presence of salts nucleo-albumin in small amount is apparently rendered more soluble by heat. A small amount shaken with distilled water, left, after a single filtration, a cloudy solution. Boiling at once cleared the latter.

[3] Save to trichloracetic acid.

[4] Medical News, May 5, 1891. That mingling of reagent and urine is avoided when the contact method is employed, as is so likely to occur by application of the flame directly to the tube.

I was then unaware of the soluble effects of high heat upon traces of this mucoid body, accepting, as is commonly taught, that nucleo-albumin reacts to heat very much as does serum-albumin.[1] It evidently requires a temperature at or quite near 212° F., and prolonged—some minutes—contact to insure a partial solution even with small percentages of nucleo-albumin. This is especially the case with trichloracetic acid. Nitric acid, and especially acidulated picric acid solution, likewise retains a certain amount of the precipitant. With unacidulated picric acid the soluble effect of heat is more decided. The contact coagulum obtained with it is dissipated to a greater degree by the aid of heat than with the others.

With potassium ferrocyanide and acetic acid, and especially with the metaphosphoric acid test, however small the percentage of nucleo-albumin present, the soluble effect of heat is not evident. On the contrary, with the latter, heat tends to intensify the reaction obtained, so that it cannot in any manner be distinguished from serum-albumin. For this reason the clinical use of these reagents as precipitants of albumin must be regarded as absolutely untrustworthy, and should be unqualifiedly condemned.

It is important to study the effects of heat upon unprecipitated urine solutions of nucleo-albumin. Heat, in any proportion of nucleo-albumin (in undiluted acid urine of ordinary gravity), upward of 0.15 per cent., is totally without precipitant effect. A few drops of dilute acetic or other acid which, added at ordinary temperature, causes a sharp cloud, are without precipitating effect upon the boiling urine. As cooling occurs, clouding results. When nucleo-albumin is present in proportion of 0.3 to 0.4 per cent., whether ebullition was previously employed in obtaining the solution or not, clouding always resulted without the addition of acid on boiling. This behavior of the various strengths of nucleo-albumin, with urine

[1] Treatises on urinalysis, save Neubauer and Vogel, state that *mucin* is precipitable by heat. Reisser, quoted by Neubauer and Vogel, found that ebullition does not change urine rich in nucleo-albumin. (By rich, Reisser probably meant upward of 0.1 per cent., which is the greatest amount he at any time encountered.)

as a menstruum, was invariable in a large number of experiments. It shows that when nucleo-albumin is present in amount approaching saturation, heat exerts a totally different effect from what it does on solutions of moderate strength. As these latter (upward of 0.15 per cent.) are, perhaps, even greater than can occur in any condition leading to the presence of nucleo-albumin in the urine, apart from a bad vesical catarrh, which cannot mislead, heat becomes the most important mode of differentiating a nucleo-albuminuria from a serumuria.

From these results it would follow that the only reliable test for serum-albumin, and that freest from liability to error, is the old-fashioned one by ebullition. But, apart from commonly recognized fallacies attending its employment, easily overcome, at least two important sources of error strikingly developed in these experiments, are likely to arise with urine containing more than traces of nucleo-albumin. Treatises on urine testing advise, in the employment of the heat test, the precaution that, should no reaction be apparent immediately on ebullition, the tube be inspected, after standing and cooling, for cloud or precipitate, which, not being found to be due to acid urates, indicates albumin or an albumose. Now, since heat is without precipitating effect upon a naturally acid, nucleo-albuminous urine, to which no further addition of acid has been made in process of testing, precipitation of the mucoid body, under the circumstances, would not occur; but if an acid be added in the application of the test, clouding will surely result as the urine cools, unless the acid has been added in too small an amount to overcome the neutralizing effects of the urinary salts. As related, the experiments with mucoid nucleo-albumin artificially added in moderate amounts to urine, indicate that a strong heat tends to dissipate the cloud produced by acids, although, after forming, it will not entirely remove all its traces.

A second source of error is that relating to the presence of excess of earthy phosphates in a decidedly nucleo-albuminous urine. In the various experiments made with mucoid-fortified

urines, specimens of the morning urines were selected. On two occasions specimens of urine voided during the day were also employed as diluents, which, like the morning specimens, were found not to react to trichloracetic acid within five minutes' contact in the cold. With these latter, and with one specimen of morning urine, although all were acid, a cloud occurred in the fortified nucleo-albuminous specimens in the heated upper portion, not dissipated by the addition of a few drops of 25 per cent. acetic acid to 10 c.c. of the boiling urine. Control specimens were found to also cloud on heating, but were readily cleared by a small amount of acid. This indicates that the presence of nucleo-albumin in a urine rich in phosphates—a combination quite usual in the healthy in the day urine, especially in that after food and exercise[1]—interferes with the solution of the precipitated phosphates by acetic acid, heat being coincidently applied to assist in the clearing of the mucoid reaction. This is an important point, ignorance of which has doubtless often given rise to error. It is of signal interest since many urine examinations, more especially those of life insurance, are made of specimens voided under conditions giving rise to the presence of excess of urinary mucin and of phosphates. But mucoid nucleo-albumin present in amount likely to originate error, reacts to acetic[2] or to citric acid, clouding more or less markedly at ordinary temperature, so that, by this means at least, the presence of excess is proved.[3] In testing for serum-albumin, should ebullition now not clear, or at least not greatly diminish, the precipitate, the excess of earthy phos-

[1] Exercise and the process of digestion tend to increase the amount of mucoid substance in the urine. From the merest trace, undetectable in the morning urine, it may rise in amount to cause a reaction to the contact nitric acid test. I have noted this in the urines of several healthy male adults since examining into this point.

[2] In the phosphatic urine mentioned, an amount of nucleo-albumin was added approximating 0.1 per cent.

[3] I have elsewhere dwelt upon the precautions necessary to insure a mucoid response with acetic acid in urines in which recognizable traces of mucoid nucleo-albumin are naturally present, with varying amounts of salts. The precipitating effect of acetic acid on urates must be borne in mind. These are differentiated by the fact that the precipitate of the latter is colored and has a characteristic microscopic appearance. They may be also recognized by the fact that a gentle heat, short of that necessary to diminish a mucinous reaction, clears the urate precipitate. Dilution of the urine, before the addition of acetic acid, will also tend to obviate a urate precipitate and tend to increase a mucoid one.

phates in a second portion of the urine should be removed in the usual manner, and heat later applied to the acid urine which has been first cleared by filtration.

It is of interest to here briefly detail the subsequent behavior of one of these albumin-free phosphatic urines to which about 0.2 per cent. of nucleo-albumin was first added, after the tendency to the marked precipitation of phosphates by heat was noted. The excess of the latter was precipitated by ammonia and removed by filtration. Later, the urine was rendered rather markedly acid and the nucleo-albumin thus thrown down removed. Repeated filtration gave a very clear solution, of a specific gravity of 1025. This, at ordinary temperature, still gave a cloud to acetic acid, but, however, tended to become quite clear on ebullition. Specimens gave a contact cloud with nitric acid and a marked contact ring with Millard's reagent; with trichloracetic acid the reaction was very decided; with metaphosphoric acid it appeared within a minute and was marked; with unacidulated picric acid solution no immediate reaction occurred, but at the end of a minute a contact haze was discernible, not increasing to an outspoken ring after standing a long time. With citrated picric acid solution a fine sharp ring was instantaneous. With all of these reagents, save the metaphosphoric acid, the extent of reaction was diminished by the application of decided heat, although with trichloracetic acid this reduction in amount of response was less manifest than with the others. The reaction of this specimen to the various tests was most instructive, as it contained a very small amount of the mucoid body, calculated by similar response of other specimens, to which measured amounts were added, to be 0.02 per cent. or less. This specimen was of especial interest for study because of correspondence in point of response to several specimens of day urines from healthy subjects I had recently examined.[1] These gave a pronounced acetic and citric acid mucoid cloud, and responded quite markedly to the delicate tests for albumin, gave a narrow

[1] Tested in all points similarly to the reinforced mucoid urine save as regards the dissolving effects of strong heat.

cloud at or near the meeting-line with HNO_3 by the contact method, but did not react to heat alone.

So far as I am aware, the valuable aid heat may render toward promoting a response, in the application of tests used by the contact method, for the detection of serum-albumin is nowhere mentioned. Since apparently heat also becomes a factor of importance in the separation of the so-called mucin reaction from that due to serum-albumin its employment is most essential where there exists the slightest doubt as to differentiation. I have frequently been able to bring out a serum-albumin reaction with HNO_3 used by the contact method, by placing the test-tube in hot water for some moments, which response otherwise was most tardy in appearing, if eventually noted at all. The use of heat in this way, rather than the application of the naked flame to the tube, is essential, that actual ebullition in the tube be not permitted, otherwise disturbance of the relative relation of reagent and urine would interfere with the occurrence of a sharp contact ring.[1]

Although I think it may be confidently asserted that a reaction so induced, as with one also apparent without heat, if decidedly sharpened by suspension of the tube in the water-bath, is serum-albumin (or serum-globulin), yet, on the other hand, since heat will not entirely remove a mucoid response after it has once appeared, there can be no great certainty as to the absence of recognizable traces of serum-albumin in the presence of the nucleoid body. If smaller amounts of albumin are looked for the same must be said of the use of the delicate tests, although it would appear if these[2] are applied with reagent and urine at the boiling-point—a method, of course, impracticable in ordinary routine urine testing—no mucoid reaction will occur so long as these are maintained at a high heat. Of the many delicate reagents, unacidulated solution of picric acid has the least reacting power to traces of nucleo-albumin. Decided preference should, therefore, be shown it over all of the others, if the employment of a refined test seems

[1] By this method acid urates and a mucoid reaction are both prevented.

[2] Excepting metaphosphoric acid.

essential. Citrated or otherwise acidulated picric acid should never be employed. Especially should it be avoided in the quantitative estimation of albumin by Esbach's method.

A serious fallacy attending the influence of heat upon the trichloracetic acid reaction in the experiments with nucleo-albumin should be here mentioned. It tends to further illustrate forcibly the impracticable extraordinary delicacy of this reagent as a precipitant of albumin and its inutility for clinical uses. In the course of the investigation a control specimen of acid urine previously unfiltered, of that portion used for experiment which gave no reaction at ordinary temperature with trichloracetic acid until some minutes' contact,[1] was thoroughly boiled and filtered while hot. This was done with the object of obtaining a control solution in all particulars similar (save as regards the presence of nucleo-albumin) to the fortified nucleo-albumin specimen. To my surprise, it was found that this boiled and subsequently but once filtered control specimen reacted more markedly to trichloracetic acid than did the unboiled, unfiltered, original specimen. I then found that both cold and hot distilled water ran once or twice through specimens of all of the various makes of white filter-paper in my laboratory, gave either instantly or after the lapse of a few moments the contact white ring with trichloracetic acid.

Messrs. Queen & Co. then supplied me with specimens of the very best grades of some of their fine filter-paper other than those which I had. Some of these they thought would certainly not so react. Bearing as this matter does upon the subject of fallacies attending albumin testing, the result is worth mention. The specimens, four in number, were marked *Swedish*, *French*, No. 390, and No. 589. They were of equal size, with a capacity of about 20 c.c. Two of each variety were used, one for hot distilled water, the other for distilled water at room temperature; 5 to 7 c.c. of each were passed through the filters in the usual manner several times. No

[1] And then but a very fine white ring, showing rather less than the ordinary normal traces of albumin.

pressure was exerted to force the water through. The distilled water in each instance was free from trace of cloud after filtration as before.[1] A slight contact white ring characteristic of traces of albumin occurred instantly with all of the specimens of distilled water filtered while hot, and tested in all, save with the specimen of French paper, while still warm. The albumin reaction was quite decided with paper No. 390. With the cold, filtered, distilled water the reaction was instantaneous only in two, with but one filtration; in the others it appeared in from one-half to one minute. In all, the reaction became very much more decided on standing a short time; but although the use of the water-bath had no diminishing effect, heat did not seem to noticeably increase the reaction, as is the case with serum-albumin. A fine cloudy haze throughout the body of the tube was, however, developed by heat, indicating a precipitating effect of the trichloracetic acid, aided by heat, on the minute traces of albumin present. Experiments of various other sorts were tried with these solutions and trichloracetic acid, which, though interesting, cannot be detailed here. Millard's reagent, plain or citrated picric acid solution, and the reagents of tartrated mercuric chloride, were found to give no indication of reaction, even after standing in contact for some time.

Millard, a number of years ago, called attention to the fact that the coarse French gray filter-paper, which contains a great deal of cellulose or vegetable albumin, would yield sufficient to react to his reagent. This I long ago confirmed, finding that several of the delicate tests reacted to distilled water passed through the gray paper.[2] Trials were made at that time with the white Swedish, French, and German makes, and the same tests were resultless, so that it had not been suspected that a fallacy from this source, with a good quality of filter, existed.

To remove the traces of nucleo-albumin from suspension it was necessary to repeatedly filter its menstruum; three to six

[1] For the sake of exactness, a control unfiltered specimen of distilled water was also tested with trichloracetic acid. As might be expected, it gave no reaction.

[2] Trichloracetic acid had not then been proposed as a test for albumin, so was not used.

papers were always employed, all of which probably contributed their quota of albumin to the filtrate, which would increase the amount of albumin recognizable by trichloracetic acid uninfluenced by heat.

Trichloracetic acid as a reagent for the detection of albumin is of such exceeding delicacy that, though evidently of little practical utility for clinical uses, it apparently may be utilized to demonstrate, without need for concentration of urine, the existence of a constant normal albumin trace. A physiological albuminuria asserted to exist by Leube,[1] Fürbringer,[2] Kleudgen,[3] Senator,[4] Chateaubourg,[5] Capitan,[6] Posner,[7] and others, has been as strongly questioned by v. Noorden,[8] Millard,[9] Lecorché and Talamon,[10] and many others. The confines of this paper do not admit of even a cursory survey of existing data for or against this important question; the subject is one that requires much space for its consideration, and will receive attention subsequently. Suffice it here to state that much of the work was done with concentrated urines, and the question of the frequent presence normally of extra-renal mucoid nucleo-albumin was often altogether ignored by those favoring the existence of a physiological albuminuria. Leaving out of consideration the frequent high percentage of marked albuminuries among the apparently healthy, to be accounted for as a result of digestive derangements, circulatory weakness, and the like, not omitting a latent nephritis, the fact that many of the experimenters in the field of physiological albuminuria, who confidently hold to the existence of a normal albumina minima, fail to consider the presence of nucleo-albumin as a complicating factor, has thrown much doubt upon the results arrived at. Still, regarding albuminous bodies other than serum-albumin, which may be present in the urine and are calculated

[1] Virchow's Archiv, 1878, lxxii. 145.
[2] Zeitschr. f. klin. Med., 1880, i. 346.
[3] Arch. f. Psych., 1881, xi. 5, 478.
[4] Die Albuminurie. Berlin, 1882.
[5] Thèse de Paris sur l'Albuminurie Physiologique. Paris, 1883.
[6] Thèse de Paris sur Albuminuries Transitories. Paris, 1883.
[7] Berliner klin. Woch., 1885, xxii. 654; Virchow's Arch., 1886, civ. 497.
[8] Deutsches Arch. f. klin. Med., 1886, xxxviii.
[9] Bright's Disease. New York, 1892.
[10] Traité de l'Albuminurie et du Mal. de Bright. Paris, 1888.

to mislead, the experiments of many observers[1] have at least shown that at times no reaction may be obtained to such delicate reagents as Tanret's and Millard's; and Leube failed to get an albumin response in some entirely healthy urines which had been subjected to much concentration. If the experiments which I have detailed with the reactions of mucoid nucleo-albumin to certain of the tests for albumin represent those obtainable when this body is present in traces in healthy urine, as I think there can be little doubt, then it can be proved that Posner is right in asserting that all urines contain demonstrable traces of albumin, and that a physiological constant minute albuminuria exists, an albuminuria which apparently is not due entirely to the presence of a mucoid body, although it may perhaps still be accidental or contingent, not arising from the blood-serum of the renal vessels.

In the paper on fallacies attending the employment of delicate tests for albumin, it was stated that out of a series of 105 urines from presumably healthy male adults, 102 gave at ordinary temperatures a contact reaction characteristic of albumin within five minutes, and that the remaining 3 also readily reacted within this time on the employment of heat, showing a contact white ring of one or more lines in depth. The reaction in the 102, further tested by heat, was apparently increased.[2]

Since the series of 105 consecutive urine examinations, all of which specimens reacted, as stated, to trichloracetic acid, I have examined a series of 35 morning specimens from presumably healthy males, in the prime of life, none of whom contributed any of the preceding specimens examined.[3] With the exception

[1] Lecorché, Talamon, Millard, and others.

[2] As the influence of heat upon a slight mucoid reaction was not then understood, less attention was paid to long contact in hot water, nor was the latter always maintained at the boiling-point, it being more especially desired to differentiate as to urates rather than as to varieties of albumin. The nucleo-albumin experiments show that unless the heat be decided, and the amount of nucleo-albumin present small, the soluble effects of the former are not apparent. In fact, although lessening of reaction with heat was quite constant with traces of nucleo-albumin, it was not always so to the same degree. It was common, too, for a mucoid response to become again decided as the temperature of the water fell from the boiling-point to a degree of heat still decided to the finger.

[3] Thirty-one of these 35 were collected for me by my friend Dr. G. M. Gould, to whom I am under deep obligations for the same.

of but 2, applying the technique detailed in the other paper, without the aid of heat, a reaction was noted in all[1] within five minutes. In nearly all of these it was quite marked in this time. In the remaining 2 a contact reaction was noted within ten minutes. In 13 the reaction was instantaneous, but in 2 only was it immediately very decided, and these were found to be examples of markedly albuminous urine reacting to coarse tests. In 6 only of the 33 could the reaction be termed slight at five minutes' contact.

The effect of heat upon all of the specimens which had reacted before its application was carefully studied, and was noted to vary somewhat with different specimens. In some it very markedly and promptly intensified the sharpness and depth of the contact ring, which had not been very decided before the use of the bath. With 4 the effect of heat was negative, viewing the tube after some minutes' exposure to the bath. The contact cloud remained as before, deepening, however, quite markedly, as was the case with all, after cooling and standing. It was certain that heat in all developed earlier a reaction which, without heat, was several minutes in becoming well-defined. In one of the 2 in which no response occurred in the cold until after five minutes' contact, it was noted that heat within a moment or so produced a contact cloud of a depth of three to four lines, which cloud supplanted the slight, although more sharply defined, white ring, evident without heat. The urine above in the tube also subjected to the bath, became hazy. On inspecting the cooled tube subsequent to the use of heat after it had been standing some time, a sharply defined contact ring was again noted of a much greater extent than at first, with a deep cloud above of some lines in depth.

One fact seems to me certain, developed by the examination of a series of nearly 150 specimens of urine from young male adults, the majority of whom are apparently in robust health,

[1] In the specimens of the 105 urines less than 10 of the number had been filtered before testing. In none of the present 35 was filtration practised. The portion to be tested was always taken from the upper stratum, after subsidence of any mucous cloud in suspension.

and none of whom had been regarded as renal suspects, that, excluding the 22 marked albuminurics, whatever the nature of the albuminous body reacting, a response may be obtained in any specimens of all urines of the healthy by the intelligent employment of trichloracetic acid. This response may not occur instantly, but usually develops within a few moments, and may be brought out in any instance by the use of heat in the manner directed.

DISCUSSION.

Dr. Judson Daland: I am sure that we all have a great desire to discover some agent by which minute quantities of albumin may be demonstrated with certainty. I think that many of these so-called delicate tests for albumin are apt to lead us into difficulty. In my own experience the discovery of minute quantities of albumin by these tests has led to the loss of hours in searching for microscopical evidences of renal change.

The observations with reference to the presence of albumin after filtering, and the possibility that this may come from the filter-paper, is exceedingly important, as is also the observations with reference to the effect of heat in bringing out more markedly the reactions with the tests which Dr. Stewart has mentioned.

Dr. J. M. Da Costa: I am more and more coming to the conclusion that delicate tests for albumin are of little use to the clinician. I believe the saying of Granger Stewart to be correct, that "if heat and nitric acid properly applied show no traces of albumin, albumin may, for practical purposes, be discarded." Picric acid does, it is true, at times give valuable information, but as a ready, yet very accurate, test, I think thorough boiling of the urine acidulated with acetic acid the best. When we publish observations, it is important to state what tests have been used. It is likely that some of the difference in results that have been reported has been due to the difference in reagents employed.

Dr. Stewart's observations are of the greater importance because they were all made on men under very similar hygienic states. I have long since come to the conclusion that the finding of small quantities of albumin in the urine of women is valueless as a means of clinical knowledge. We must trust to the microscope entirely.

It seems to me that something else will grow out of researches of this kind. Why should we not trace up this nucleo albumin and peptone and proto-peptone to its cause, and try to connect them pathologically with some change in the kidneys and other parts of the economy? Who knows but

that by studies of this nature a discovery scarcely inferior to that of Bright awaits the inquirer?

Dr. Lawrence Wolff: I have followed Dr. Stewart's experiments with a great deal of interest. There is no doubt about the delicacy of the trichloracetic acid test, and the fact that heat increases the reaction differentiates it from the reaction for the protopeptones, in which the latter dissolve on heating.

With regard to the specimens of urine used in these tests, I was myself the agent for procuring many of them from men who were studying urinology under my instruction. These men were familiar with the precautions required in securing the specimens, and had not been exerting themselves. I think that it may be positively stated that these were the best-selected specimens of urine ever experimented with. I agree with Dr. Da Costa that trichloracetic acid will not serve in the clinical examination of urine.

The researches of Dr. Stewart only confirm those of Senator, who, in his classical memoir on the subject of albuminuria, has shown that there is a trace of albumin present in all urines. It is unfortunate that Dr. Stewart should have applied the term nucleo-albumin to this substance. Nucleo-albumin in addition to albumin contains phosphorus and at times iron. Mucin and mucoids are sometimes classed with nucleo-albumin, and we speak of mucoid nucleo-albumin. In the kidney we have serum-albumin first transuded from the glomeruli into the capsule of Bowman, which mixes with the urine in the uriniferous tubules, which is free of albumin. The addition of nucleo-albumin, or the partly digested or dissolved cells of the epithelium of the ureters and bladder, only occurs in more or less marked grades of a catarrhal inflammation. I believe that the larger amount of the albumin indicated by the trichloracetic acid is not nucleo-albumin, but to a large extent serum-albumin, which we find in normal quantities present in the urine. This, however, does not detract from the value of the observations. I believe that Dr. Stewart has clearly shown that trichloracetic acid is for practical purposes not available as a test for albumin, and that albumin is present in all urines in normal conditions, as Senator had pointed out.

Dr. J. P. Crozer Griffith: I wish to say a few words because reference has been made to the test with ferrocyanide of potassium and acetic acid, and I have had considerable experience with this. I first began to use it with great confidence, but have gradually become more and more suspicious of it. I wish to add my testimony that I fear we cannot depend upon it as a test for serum albumin. It was originally held by some who recommended it, that if on the addition of acetic acid a cloudiness developed, mucus was present, and that an additional cloudiness, produced by the addition of ferrocyanide, was due to albumin. If this is true albumin is frequently present in the urine of persons whom we really cannot regard as sick.

I am glad that Dr. Da Costa has referred to the difficulties experienced in the examination of the urine of women. In practice it seems largely for-

gotten that in the examination of the urine of women we are entirely at sea on account of the adventitious albuminuria which may be present; this being the result of the admixture of vaginal discharges. It has seemed to me that we could to a certain extent balance the chemical and the microscopical examinations. If we find a very sharply cut ring of albumin it is probable that there is some trouble in the kidney; whereas, if there is a thick but not sharply defined ring of albumin, and the microscope shows a large number of pus cells, it is probable that the albumin is due to the presence of pus. This is a coarse method, and if Dr. Stewart can give us a finer way of making the diagnosis we should be glad to have it. I hope greatly that he intends to continue his studies in this direction.

Dr. H. C. Wood: I should like to ask a question in regard to albuminuria in women. The position which has been taken is not entirely in accord with my own experience. I believe myself that you can never accept the results of the examination of the urine of women passed in the ordinary way. Anyone who wishes to do proper work will see that the urine is drawn with the catheter. I should like to know if under such circumstances there is any difference in the urine of women and men?

Dr. J. M. Da Costa: It is hardly possible except in hospitals to secure the urine by catheter. If we could, this would undoubtedly remove a large source of error. Whether or not it would remove all error, I cannot say, because of the great frequency of catarrhal conditions about the neck of the bladder in women.

Dr. S. Weir Mitchell: It may be interesting to recall Dr. Keen's case in which the urine showed no albuminuria unless the man took active exercise afoot. As long as he was quiet in bed no albumin could be detected. It was worthy of note that the most severe massage brought on no albuminuric condition, thus showing a very material difference between passive exercise and that obtained by voluntary exertion.

Dr. Stewart: As I have distinctly stated in my paper, its object was not an inquiry into the existence of a physiological albuminuria. The investigation was on lines that Senator has not entered into at all in his book referred to by Dr. Wolff. A neglect to consider the question of the reactions of so called mucin with the commonly employed albumin tests has laid the value of much of the work of Senator open to question. I have shown that so-called urinary mucin reacts similarly in many particulars to the peptones as well as to other albuminous bodies occurring in the urine, and as mucin often exists in some quantity in normal urine it must be regarded, or the results obtained with certain tests will certainly mislead.

My paper, as the title plainly states, is an investigation of the reactions of this body falsely styled mucin, which so often exists in some quantity in the day urine from normal individuals, with the commonly employed tests used for the detection of serum-albumin. I believe there is no doubt that it has brought out several facts of considerable clinical value as regards albumin

testing. It certainly shows that a few tests, such as the trichloracetic acid test and those tests in which acetic acid or other of the vegetable acids, such as citric or tartaric are used, are of no practical value as tests for serum-albumin. As to the question of the existence of a physiological albuminuria, my experiments have elicited what neither Senator nor anyone else had before shown, that the presence of an albuminous body, in all likelihood not nucleo-albumin or mucin, but probably serum-albumin or globulin, may be shown in the urine of everyone, without a single exception, no matter when voided, by the use of the first named of these reagents—trichloracetic acid.

A NEW HÆMATOKRIT AND A NEW TECHNIQUE.

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[Read May 2, 1894.]

This instrument[1] is composed of a series of wheels, by which an upright spindle may be rotated 10,000 times per minute. The metal frame is placed upon a spindle, and so arranged that it carries two glass tubes, the outer end of each fits into a small cup-like depression, the bottom of which is covered with thin rubber. Each of the inner ends of these glass tubes presses against a similar surface, which is fastened to a spring, so that it may be pulled toward the centre while each tube is being introduced. So soon as the tube is in place this spring resumes its normal position, holding the tube firmly and safely. This arrangement is extremely simple and effective. The tubes are placed *between* the two arms which constitute the metal frame, and the frame, having but little thickness, cuts through the air, thus minimizing atmospheric resistance. Each tube measures fifty millimetres in length, with a lumen of half a millimetre, and upon it is a scale representing 100 equal parts, and has a lens front which, by magnifying the column of blood, facilitates and renders more accurate the reading of the scale. One end of each tube has a blunt point, somewhat like that seen in the Thoma-Zeiss hæmocytometer. A single revolution of the large handle produces 134 revo-

[1] This instrument is made by F. F. Metzger, 470 Dillwyn Street, Philadelphia, U. S. A.

lutions of the frame, so that to secure 10,000 rotations per minute the large handle must make 77 revolutions. This question of speed is important, and, when 10,000 rotations are secured in *one minute*, a compact volume of blood-cells is formed which does not permit itself to be still further reduced, even though rotation be continued for several minutes.

The instrument should be firmly secured to a solid table, and when in active use should be thoroughly oiled once daily. The method of using this instrument is simple. To fill the glass tube, a rubber tube is slipped over the end of the capillary pipette, and to the extremity of this rubber tube is attached a mouthpiece, precisely in the same manner as when the hæmocytometer is employed. This glass tube, or pipette must be absolutely *clean* and *dry*. The finger of the patient is punctured; the blunt point of the pipette is then placed into the blood, and the tube completely filled by suction. The finger of the operator is quickly applied to the blunt extremity of the tube, which is then inserted into the frame, and rotated at a rate of 10,000 times per minute. All that remains is to read the percentage volume of blood from the scale. The divisions on the pipette are one-half millimetre apart, so that one may read this scale without difficulty. The entire procedure need not occupy more than three minutes, and requires no special nor unusual skill, while the elements of error are so few that the results are trustworthy, even when performed during the rush and hurry of clinical work.

Although Professor Blix was the first to suggest the use of centrifugal force in the separation of red and white blood-cells, this hæmatokrit presents many features that are original, and the technique is new. The mechanism by which this extraordinary velocity is obtained is simple, compact, and is protected from dust and injury; further, the number of revolutions per minute is greater than has ever been hitherto secured. This high rate of speed requires the expenditure of but little power, as the handle is turned with ease, and the apparatus is so adjusted and balanced that the chief force to be overcome is that offered by the atmosphere to the rapidly revolving frame. The

capillary tube possesses the advantage of increased length, and a scale of 100, each division of which represents half a millimetre, with a lumen of the same diameter. The amount of blood required is no more than that employed in making a blood count. The advantages possessed by a scale divided into 100 parts are manifest, inasmuch as the volumetric per cent. of corpuscles may be at once determined without multiplication.

In extreme anæmias, as well as as under other circumstances the practical importance of using but a small quantity of blood should not be under-estimated. In these conditions the amount of blood obtainable from an ordinary puncture is frequently limited, unless one resorts to gentle massage, which disturbs the normal relations existing between the corpuscles and the plasma. In practice one is able, without difficulty, to prepare a fresh specimen of blood for microscopical examination, and make a hæmoglobin and hæmatokritic estimate, from the blood emerging from one puncture, and these procedures may be all performed before coagulation occurs. The conversion of one end of the glass tube into a blunt point, facilitating its filling, is a convenience that will be greatly appreciated by workers in this field, while the ease with which the tube may be cleansed is no small advantage when one remembers the difficulty often encountered in the cleansing of the Thoma-Zeiss hæmocytometer. To facilitate comparison, it is desirable that the results obtained from the various examinations of the blood should be expressed in percentages, as is the custom in reference to hæmoglobin. With this end in view, 5,000,000 red blood-cells per cubic millimetre may be recognized as normal or the equivalent of 100 per cent. As the normal volume of blood-cells is approximately 50, it may be considered arbitrarily as normal or 100 per cent.[1] To secure this result it is necessary to double the number as read from the scale of the tube.[2] This simple method of expression of the results of the examinations of the blood by the hæmatokrit enables one to determine, with a fair degree of accuracy, the proportion exist-

[1] The exact figures are 51.6 per cent. of blood-cells and 48.4 per cent. of plasma.

[2] See Fortschritte der Medicin, November 1, 1891. No. 21, page 869.

ing between the volume of corpuscles and the amount of hæmoglobin present, which, in its turn, gives the proportion that probably exists in each individual red blood-cell. For illustration it may be stated that, if the hæmoglobin was 50 per cent., and the hæmatokrit showed 100 per cent., the proportion of hæmoglobin in the corpuscles would be $\frac{50}{100}$, or one-half, *i. e.*, the hæmoglobin contents of each red cell is reduced one-half. This estimate of the amount of hæmaglobin in each corpuscle is of considerable diagnostic and therapeutic importance.

The use of the hæmocytometer requires much care, labor and time, and an experience of several years has convinced me that, under ordinary conditions, the counts obtained are often far from accurate. In 1891 a series of experiments were made to devise a method that would give more accurate results with the expenditure of less time, labor and skill. The results of these experiments were incorporated in a paper,[1] since which time additional observations have been made, of which the following is a summary:

The chief objections to the hæmocytometer are the difficulty of estimating the quantity of diluted blood to be placed upon the slide; of placing the top cover in proper position; of securing an even distribution of the corpuscles over the field; of obtaining a complete mixture of the blood with the diluent, as there is but one part of blood to 100 parts of the diluting fluid; the eye-strain and nervous exhaustion consequent upon counting the large number of squares necessary to secure accurate results, and, finally, the very considerable expenditure of time. Numerous experiments were made, and a difference of from 187,500 to 525,000 or more was observed, even though the blood was examined from the same drop by two skilled independent observers.

Heretofore all investigators have believed it necessary to dilute the blood with a liquid that would prevent coagulation prior to subjecting it to centrifugal force, but a large number

[1] Fortschritte der Medicin, October 15, 1891, No. 20; and Fortschritte der Medicin, November 1, 1891, No. 21.

of experiments have proven that blood may be drawn directly into the tube, and rotated immediately thereafter, before coagulation is possible. Moreover, repeated observations have shown that when the blood is rotated rapidly 10,000 times the volumetric per cent. thus obtained is not reduced by continuing the rotation. As this rotation of the frame may be secured in one minute, and the blood drawn into the tube in another minute, the entire procedure, under favorable circumstances, need not occupy more than two to three minutes.

In view of the fact that so great a centrifugal force is employed, the question naturally occurred as to whether or not the corpuscles were injured thereby. To determine this question a number of microscopical examinations were made of the corpuscles after they had been rotated, and it was observed that they presented a normal appearance. Heretofore it has been rather difficult to obtain the plasma unchanged and transparent so that spectroscopical examinations could be made, but with the hæmatokrit the plasma, remaining in the proximal extremity, lends itself readily and easily to this examination, and thus opens a field of study regarding the chemical condition of the plasma, as shown by the spectroscope, which may yield valuable results.

An examination of fifty-five healthy males showed an average volumetric per cent. of 103, and a similar examination of eight female nurses gave an average of 88 per cent. This latter figure is probably less than the true percentage, as most of these women were somewhat anæmic. When the blood corpuscles in the blood of twenty-five healthy males were counted it was found that each volumetric per cent. was practically the equivalent of 50,000 red blood-cells, so that, to convert the volumetric percentage into the probable number of red corpuscles present, it was only necessary to multiply this per cent. by 50,000.

Prior to the discovery that it was possible to draw blood directly into the tube, the method adopted was to dilute the blood with an equal quantity of 2.5 per cent. solution of bichromate of potassium. This solution the author discovered

after a series of experiments with nineteen different solutions, and it not only acted as a diluent, preserving the normal appearance and shape of the cells, but it absolutely prevented coagulation. Even with this old technique, a prolonged series of observations upon a healthy male showed that a variation of but from 2 to 4 per cent. occurred. The principle sources of error with the old technique and Hedin's hæmatokrit[1] were:

1. The difficulty in measuring and correctly diluting the blood.

2. The error occasioned by evaporation, which quickly takes place so soon as the blood is exposed to the air.

3. The numerous air bubbles that form as the blood is mixed with the diluting fluid.

4. The tendency of the red blood-corpuscles to gravitate to the bottom before they were drawn into the tube of the hæmatokrit.

5. The shortness of the tube, having a scale of but 50, and the dilution by the solution of bichromate of potassium necessitated multiplication by four.

These difficulties have been obviated (1) by drawing the blood directly into the capillary tube, and (2) by the modification of the tube as regards its length and the divisions upon the scale.

The volumetric estimate of the blood is so readily performed by this new hæmatokrit, combined with the new technique, that this instrument may now be employed, not only in clinical research, but in ordinary practice. The errors to which this instrument is susceptible should not exceed 1 per cent.

As each white corpuscle probably occupies a larger space than one red cell, and as one division of the scale, which measures a half millimetre, is the eqivalent of 100,000, it is evident that this space, if it were filled with white corpuscles would probably contain a less number. By the aid of a small magnifying glass it is possible to estimate one-quarter of this space, but the recognition of a *slight* leucocytosis is practically

[1] Hedin: Scandinavisches Archiv für Physiologie, 1890, No. 2, 134; Prager medicinische Wochenschrift, 1891.

impossible. When the increase in the white blood-cells rises to 30,000 or more this grade of leucocytosis is readily detected. When a column of red corpuscles obtained from a normal individual is examined, the white blood corpuscles present a sharp, clearly defined and very shallow white band. When the leucocytes are diminished in number this white band is imperfect, and, in places, the red color of the bi-concave disks is visible.

Experiments are now in progress to determine its actual value in hospital work, the results of which shall appear in a subsequent communication.

DISCUSSION.

Dr. Frederick P. Henry: I have been greatly interested in the exhibition of this instrument, which Dr. Daland has so modified as to make practically his own, and it is very gratifying to me to witness the growing interest in hæmatology, especially in its clinical applications. Prof. Hayem closes his great work on the blood with the sentence: "The future belongs to hæmatology."[1] It seems to me that the present has claimed this science as its own. I have seen it evolve from very humble beginnings. My interest in the clinical examination of the blood dates from the publication of a paper by Dr. E. L. Keyes, of New York, on mercury in the treatment of syphilis.[2] This was an epoch-making paper, and yet from our present standpoint, it is very faulty. Dr. Keyes endeavored to prove that mercury, at least in the treatment of syphilis, is a hæmatinic. He did prove that the red blood-corpuscles of a syphilitic patient under mercurial treatment increase in number, but he made no observations concerning their value. At the time of his investigations the importance of the color test was not generally recognized. The same criticism applies to a paper of my own.[3] I demonstrated a diurnal variation in the number of the red corpuscles, and showed that this coincided with diurnal fluctuations of the body temperature. I believe my observations to be of physiological value, but they would be more valuable if they contained, in addition to a count of the corpuscles, an estimation of their percentage of hæmoglobin.

Before I became acquainted with the color tests of Gowers and Fleischl I had become aware of the fact that a mere count of the red and white cor-

[1] l'Avenir appartient à l'Hématologie.

[2] Am. Journ. Med. Sci., Jan. 1876.

[3] Cartwright Essay, 1881.

puscles was a very partial examination of the blood, for I was constantly meeting with patients, especially of the female sex, who, with all the symptoms and signs of anæmia, had a normal number of red corpuscles. There is another method of examination which has lately come into vogue, and which is indispensable to a thorough clinical study of certain affections, and especially of leukæmia. I refer to the staining methods introduced by Weigert. We now know that it is not sufficient, in a case of leukæmia, to determine that the white cells are increased in number. We must determine, besides, in what proportion the different varieties of leucocytes are increased. Only those who have attempted to do this can appreciate the difficulties in the way of its accomplishment. In fact, the thorough study of a case of leukæmia can scarcely be called clinical, for it must be pursued in the laboratory rather than at the bedside.

Another method of examination has been introduced by Henoch, of Paris. It is the function of the red corpuscle not only to carry oxygen to the tissues, but to deliver it up to them. If this delivery does not take place, in other words, if the oxyhæmoglobin is not converted in a given time into reduced hæmoglobin, the function of the red cells is not properly performed. By means of his hæmatoscope[1] Henoch claims to be able to determine the period of this conversion. I am not aware that the claims of Henoch have been confirmed, but it cannot be denied that the object of his instrument is an important one.

Dr. J. K. Mitchell[2] has recently demonstrated that the number of red corpuscles in the superficial vessels is greatly increased by massage. His suggestive paper raises the question to what extent the red corpuscles of a normal specimen of blood (normal as to number and color) are in active function. An instrument to determine this point is a desideratum.

I have said enough to show that a thorough examination of the blood cannot be made by any single instrument. With the hæmotokrit the percentage of number can be ascertained more speedily and probably with greater accuracy than with any of the counting instruments; and this, I believe, is all that is claimed for it. Even with these limitations, it is a valuable addition to our methods of investigation.

Dr. J. M. Da Costa: Dr. Daland's instrument seems likely to prove of great value. It is utterly impossible to introduce an instrument into general use that requires any length of time for its manipulation. Such an instrument remains in the hands of the few and in laboratories. The great advantage of the instrument shown is its easy applicability. It will not enable us to do without finer research, and especially microscopical research; but it will be an immense aid to the clinician, and will let the practitioner at large share in the scientific work that is going on. That it will be a great credit to the inventor I feel convinced.

[1] Exhibited and described by me in 1889, Trans. Coll. of Phys., Philadelphia, vol. xi.

[2] Am. Journ. Med. Sci., May, 1894.

Dr. Alexander C. Abbott: An advantage of Dr. Daland's instrument that has not been brought out in the discussion is, that it does away with the personal element present in counting by the Zeiss-Thoma and Gower instruments. This instrument is a step in the direction of introducing precise methods into this work. Too much should not be expected of it. We cannot expect any single method or instrument to afford a complete analysis of a composite body. This instrument only indicates the volume of corpuscles present, but it does this exactly; and I consider it a decided addition to our armamentarium.

Dr. Daland: A complete examination of the blood necessitates the determination of the specific gravity, the enumeration of the number of white blood cells, and the estimation of the hæmoglobin, all of which require special instruments. Nor can we dispense with the valuable information gained by the reaction of the blood as well as the various aniline dyes. The hæmatokrit is rather a rival of the blood-counting method than of any other, and proposes to substitute the volumetric for the numerical method. However, the instrument has advantages in other directions; in the examination for tube casts in urine; the estimation of the fat per cent. in milk; the examination of the liquids from serous sacs, the result of inflammatory changes, and the examination of liquefied sputum and other liquids in which tubercle bacilli are supposed to exist. The very high rate of speed that may be produced by this instrument, and the consequent increase of centrifugal force, opens up a very promising field for investigation in these several directions.

NOTE IN REGARD TO FRACTURES OF THE HUMERUS AT THE ELBOW-JOINT.

By CHARLES W. DULLES, M.D.

[Read June 6, 1894.]

The treatment of fractures at the lower end of the humerus is one of the well-recognized difficulties in surgery. The reason for this is chiefly the complicated character of the elbow-joint, the number of bones entering into it, the anatomical features of the joint, and the disturbance of function which results from slight displacements. The danger of permanent stiffness of the joint used to be regarded as one of the most serious features of these cases; but in recent years this danger seems to be more usually avoided, and mere stiffness is not feared as it once was. The point in regard to the treatment of these fractures which now attracts most attention is the very important one of preserving the normal inclination of the plane of the joint surface of the humerus to that of the principal axis of its shaft. This forms on the radial side a moderately acute, and on the ulnar side a moderately obtuse angle, resulting in a slight angle between the axis of the forearm and of the upper arm in the position of extension, which contributes very materially to the usefulness of the arm for a variety of purposes, and especially for carrying weights in the hand. The importance of this has been frequently and strongly emphasized of late years, and has to a certain extent dominated the treatment of injuries near the elbow-joint. Another source of trouble in fractures at the lower end of the humerus, to which as much attention should be paid, is rotary displace-

ment of the fragment or fragments of the humerus. The result of this displacement is sometimes exceedingly unfortunate, the external condyle, for example, being displaced in a direction forward and toward the middle line (ventrad), producing a deformity which is not uncommon, and which destroys what is called the "carrying-angle" as effectually as the more commonly recognized upward displacement of the internal condyle, because it destroys the normal relation of the radius to the humerus and to the ulna, and materially restricts the motion of supination.

To understand the condition, involved in fractures at the lower end of the humerus, it is necessary to study the normal mechanical conditions of this joint. Its complicated character makes verbal description almost impossible; but certain points, easily demonstrable from the bones, ought to be understood and remembered.

The first of these is the well-known obliquity of the plane of the joint, by which the really articulating bone—the ulna—in a position of extension of the forearm, with the arm hanging at the side, is directed somewhat away from the axis of the body, so that the elbow can get a purchase against the hip, and objects carried in the hand do not touch the leg; while, on flexion, the hand is carried across the line of the axis of the humerus in the direction of the chest in a manner which has a number of practical advantages. Another point which I have not yet found referred to in any work on surgery, general or special, but which is familiar to observant anatomists is, that the head of the radius, when the forearm is extended, is not in contact with the capitellum—the articular surface of the external condyle of the humerus. Freedom of the motions of pronation and supination is favored by this fact, and at the same time the joint is to a certain extent protected from accident. With the arm extended and the forearm strongly pronated, the head of the radius is at its greatest remove from the capitellum. With the forearm semi-flexed and semi-pronated, the head of the radius rests against the capitellum; while if flexion of the forearm be carried further, there occurs a con-

dition which I have taken this occasion to describe particularly. I do not know that it has been as yet described, although I know that the conclusion to which a study of it has led me coincides with what has been done before, and I have been interested to ascertain by a correspondence accidentally started, that Dr. H. L. Smith, of Boston, has been brought to similar conclusions by studies undertaken before he knew that I was working on this subject and by actual practice. The point I wish to make is, that when the forearm is semi-prone and fully flexed—that is, until flexion is stopped by the impact of the coronoid process against the humerus in the coronoid fossa—the bones of the forearm act as a lock to hold the lower end of the humerus, in case of an intra-condyloid fracture, in a correct anatomical position, and that in no other position of the forearm is this the case. If we take the bones in this position we find that the olecranon and coronoid processes, with the ridge dividing the greater sigmoid cavity, control the inner part of the elbow-joint, preventing motion laterally, antero-posteriorly, or rotary, while the head of the radius rests against the capitellum, and its ligaments prevent displacement of the external condyle in any direction by the condition of tension in which they are found when the forearm is fully flexed and semi-pronated. To say that the bones of the forearm lock the inner and outer portions of the lower end of the humerus in a correct anatomical position, covers the essential points in the treatment of a fracture; and to understand the full significance of the expression requires only a knowledge of the anatomy of the elbow-joint.

As I wish to be brief, I will limit myself to this statement, adding only a particular reference to the obliquity of the plane of the joint and the danger of rotation of one of the fragments, as alluded to above.

To meet the indications suggested by these anatomical considerations, I apply from the fingers to the shoulder, smoothly and with moderate tension, a soft flannel bandage, avoiding undue pressure upon the anterior angle at the elbow by having the forearm flexed and semi-pronated while the bandage is ap-

plied. After this the bend at the elbow is filled with some cotton, a splint is laid against the arm with very moderate cotton padding—not bound on with a bandage, but just laid loosely on—and the whole is enclosed with a good roller bandage. This treatment I have found to be entirely comfortable to the patient, and to give admirable results.

The essential feature of the method which I propose is, that the forearm shall be semi-pronated and fully flexed, with the intention that the ulna and radius shall then hold their corresponding parts of the joint-end of the humerus in a correct anatomical relation to each other. The mode of securing the arm is a detail which need not be discussed here, as any one who treats many elbow-joint injuries knows that it is impossible to fix inflexible rules for their management.

Any splint may be used which will maintain the forearm in full flexion upon the upper arm. For this purpose a splint with a very acute angle, to fit either side of the arm, or an anterior angular splint will do. The principal difficulty in following out a theoretically correct method at the elbow-joint is due to the swelling which, in this situation, comes so quickly and often becomes so considerable. My own experience leads me to believe that it is desirable to effect reduction of this swelling by a carefully applied flannel bandage, assisted with wool or cotton, so as to produce equable pressure. This may be used for twenty-four hours before applying the splint which is to maintain the arm in the position above referred to; and while it is done the arm may be kept in almost any position that is comfortable to the patient. I find that it requires some ingenuity to neatly bandage an arm to a lateral angular splint, but no more than every surgeon may be supposed to have.

This is not the time to dwell upon details which all surgeons understand. The object of this note is to call attention to the fact, as I believe, that, if the forearm be semi-pronated and fully flexed upon the humerus, the head of the radius and the coronoid and olecranon processes of the ulna will hold the inner and outer portions of the lower end of the humerus in

a correct anatomical relation to each other and to the bones of the forearm. With all this, it is possible that a T-fracture may turn out badly; but, if it should, this would be in consequence of a displacement at the line of transverse fracture; and I believe the chances of such an accident would be diminished by securing accurate anatomical relations of the bones forming the elbow-joint.

DISCUSSION.

Dr. Addinell Hewson: I think that the points advanced by Dr. Dulles are well taken both from an anatomical and surgical standpoint. The fixation of the joint depends upon two factors—first, the contour of the bones and, second, although not of so great importance, the deltoid shape of the lateral ligaments. In the fixation of this joint Dr. Dulles brings the head of the radius in accurate approximation with the capitellum. The remaining portion of the head, together with the orbicular ligament, occupies the space immediately above the capitellum, and prevents the angle being made less than forty degrees. This angle is made greater by the position in which he places the hand, midway between supination and pronation. I think that the points that Dr. Dulles has made have been brought out in the adaptation of the Stromeyer splint to the treatment of fractures at this joint. If we recall the method of formation of the lower end of the humerus it will be remembered that the epiphysis is divided into four parts—the external condyles and internal epicondyle, the point of ossification for the trochlear surface and for the capitellum. These points of ossification vary in the time of their attachment from the third to the eighteenth year. This will make a great difference in the treatment of fractures.

I have had several cases of fracture at the elbow-joint in which I have obtained good results, although not using the treatment employed by Dr. Dulles. One case was that of a young man, aged eighteen years, with a T-fracture at the elbow-joint. In that instance there was obtained complete use of the joint and preservation of the carrying function. In this case an anterior angular splint was used, and I attribute the result to the care that was employed. The anterior splint in this case was furnished with a Stromeyer screw. In this way one splint is sufficient. The only modification that might be made would be, if the splint is to be worn in the extended position, to bring out the natural angle. The lines of the humerus and ulna are at an angle in the straight position, but in the flexed position the line of the humerus and the inner line of the ulna are in the same vertical plane.

Dr. Richard H. Harte: I have been much interested in Dr. Dulles's remarks. Looking at the anatomical relations of the joint, there is one

point that is often overlooked. If we examine the lower end of the humerus we shall find that the larger portion of the articular surface is anterior to the long axis of the bone, and that the natural functions of the hand and forearm are performed largely with the forearm at or near a right angle with the arm. Of course there are reasons why in certain conditions of fractures at the elbow the fragments may be in better position in the extended position, but, as a rule, the flexed position is the most satisfactory. In the flexed position the ligaments are relaxed almost equally, and the flexor and extensor muscles are also relaxed. With the arm in a right-angled position there is very little tendency to displacement.

Of course, as Dr. Hewson has suggested, the epiphyseal element comes in in young subjects. I think that in such case subsequent trouble is oftener due to epiphyseal separation and the changes that follow than to any special displacement of the fragments. I think that we are less liable to have interference with function in adults than in young persons.

With regard to the extended position, in the past year I have known of two cases where the arm had been so treated, and where it had been left fixed in this position, requiring subsequent resection. I think that with the anterior or internal angular splint we get as good results as with any other mode of apparatus.

CHRONIC VALVULAR DISEASE OF THE HEART.

A Study from Clinical Observations of 1024 Cases.

By THOMAS G. ASHTON, M.D.,
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[Read June 6, 1894.]

Although in the analysis of the following cases some points already well known are reiterated, the number of cases collated is so large that even a confirmation of these facts cannot fail to be of value. In addition to this, however, I trust that the report will be found to possess features that will throw additional light upon the interesting subject of which it treats. These observations were made during a period of six years in the course of examination of applicants for life insurance. I regret that I am unable to give the total number of cases examined, and thus indicate the prevalence of valvular disease in its various forms.

Statistics dealing with the relative frequency with which the different cardiac valvular lesions occur, together with the various combinations and associations of these lesions, are found to differ very much. The reason for this difference becomes obvious if we take into consideration the different conditions under which the observations are made. If these observations be made in a series of cases which have never presented so-called cardiac symptoms, and in which the finding of the cardiac disease occurred accidentally, as in the course of a routine examination, the result will be found to differ vastly from that obtained by analyzing a series of cases manifesting

cardiac symptoms, and which seek advice on account of those symptoms. In the former group of cases the predominating lesions will be found to be simple in character and unassociated with any alterations in other valve systems; in the latter there will be found to predominate those pure and associated valvular lesions that are most prone to give rise to changes in the walls of the heart, and hence cause cardiac symptoms.

Again, if we compare the results obtained under either of the conditions noted with those obtained by post-mortem examinations the difference will be yet more striking. Of course, the results obtained from post-mortem examinations are accurate as regards evidence of organic disease, but such results would not represent the frequency with which cardiac bruits are heard during life, as murmurs, and especially the murmur of mitral incompetency, occur in many cases that at the autopsy fail to present demonstrable lesions.

At the outset, therefore, it is well to understand that this report includes but few cases that presented cardiac symptoms, and that in almost every instance the cardiac lesion was discovered in individuals presumably in the best of health and themselves totally unaware of the existence of any serious disease. For this reason it will be found that the number of cases presenting combined or associated lesions is relatively small, and from this standpoint alone the study of these cases assumes additional interest as emphasizing the well-known fact that cardiac disease may exist for years and the knowledge of his condition not be forced upon the attention of the victim.

In order to present the facts of the report in as clear a manner as possible I have tabulated the cases in such a way as to show the relative frequency of the various valvular lesions, the predisposition of the sexes, the various ages at which the lesions were found, and such history of antecedent disease as it was possible to obtain and to which might be ascribed the existence of the valvular disease.

In common with other statistics, the fact is brought out in this analysis that in by far the largest percentage of cases the mitral valve was the seat of the lesion.

Thus we find that there were of cases of—

	Males.	Females.	
Mitral insufficiency	295	262	557
Mitral stenosis	15	17	32
Mitral stenosis and insufficiency combined	13	19	32
The total being . . .			621

or 60.6 per cent. of the entire number of cases under observation.

The percentage of cases showing simple mitral insufficiency is 54.4 per cent., while the lesions of mitral stenosis alone and combined with insufficiency each reached 3.13 per cent.

There were of cases of—

	Males.	Females.	
Aortic stenosis	101	35	136
Aortic insufficiency	32	15	47
Aortic stenosis and insufficiency combined	27	11	38
The total being			221

or 21.58 per cent. of the entire number.

Of combined aortic stenosis and insufficiency associated with mitral insufficiency there were 93 cases, or 9.08 per cent. of the cases collated, and of these 65 occurred in males and 28 in females.

Of aortic stenosis associated with mitral insufficiency there were 45 cases, or 4.39 per cent. of the total—31 of these occurred in males and 14 in females; while of aortic insufficiency in association with mitral insufficiency there were 33 cases, or 3.22 per cent.—26 occurring in males and 7 in females.

Insufficiency of the tricuspid valves was met with in 11 cases, or 1.07 per cent. of the whole, 7 being in males and 4 in females.

A study of the relative liability of the sexes to cardiac valvular disease, based upon the accompanying table, shows that a considerable majority of the cases occurred in males: thus, 612 men, or 59.77 per cent., were affected, while the disease was found in only 412 women, or 40.23 per cent of the whole,

although the number of each sex examined was about the same.

By lesions of the mitral valve the sexes were almost equally affected, there being in a total of 621 cases but 25 more males than females. In a total of 64 cases in which mitral stenosis, alone or combined with mitral insufficiency, was found, the relationship of the sexes was 28 men to 36 women. While this relative liability of the sexes to these lesions corresponds closely with that found by Duroziez,[1] it is at marked variance with the results obtained by Middleton,[2] who found that women are more liable to these lesions than men, the proportion being three of the former to one of the latter. With Middleton's statistics also agree those of F. J. Smith,[3] which are based upon an analysis of the records of the London Hospital for eleven years, and which show that mitral stenosis, alone and combined with incompetency, occurred in 71 women and 22 men.

To aortic disease we find that the male sex is much more liable than the female; thus, including all cases in which evidence of aortic disease existed, whether associated with mitral disease or not, there were 282 men and 110 women, a proportion the accuracy of which is corroborated by an analysis of F. J. Smith's statistics, which show that 263 cases of aortic disease occurred in men, while in only 105 cases were women the subjects of this lesion.

This difference in the relative liability of the sexes to the various valve lesions is, I think, readily explained by the fact that mitral lesions usually result from a cause to which, taking all ages together, both sexes are almost equally liable, viz., rheumatism. On the other hand, as aortic disease occurs most frequently as the result of arterial sclerosis and atheroma, and as men are more frequently the subjects of the causes, such as laborious occupations, syphilis, and alcoholism, which lead to sclerotic changes in the vessels and valves, it follows that they are more predisposed to aortic disease than women.

[1] Traité Clinique des Maladies du Cœur, Paris, 1891, p. 319.
[2] Lancet, London, October, 1889.
[3] Sansom: The Diagnosis of Diseases of the Heart and Thoracic Aorta, 1892, p. 387.

Lesion.	Sex.		Age.																Occupation.		Antecedent history.						Sclerosis or atheroma of bloodvessels.	Total.	Per cent.
	Male.	Female.	1–10		11–20		21–30		31–40		41–50		51–60		61–70		71–80		Light.	Laborious	Rheumatism.	Infectious fevers.	Syphilis	Alcoholism.	Negative.					
			M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.												
Mitral incompetency	295	262	14	12	43	69	118	54	41	29	37	31	31	40	11	2	..	...	222	334	[1] 13	54	47	93	125	169		557	54.40
Aortic stenosis	101	35	3	1	5	1	9	4	15	6	27	11	32	10	10	2	...	...	43	93	42	15	17	34	23	59		136	13.28
Aortic stenosis and incompetency, with mitral incompetency.	65	28	7	3	4	8	10	5	6	3	15	5	16	4	6	...	1	...	34	59	37	8	12	18	11	32		93	9.08
Aortic incompetency	32	15	2	3	1	1	4	2	5	1	8	4	9	3	3	1	..	.	19	28	18	4	3	10	9	13		47	4.59
Aortic stenosis and mitral incompetency.	31	14	1	...	3	1	4	1	3	2	7	3	9	4	4	2	...	1	15	30	15	3	5	13	6	17		45	4.39
Aortic stenosis and aortic incompetency.	27	11	1	...	2	1	4	...	5	2	6	2	8	6	1	...	...	...	9	29	17	4	3	5	7	15		38	3.71
Aortic incompetency and mitral incompetency.	26	7	2	1	2	2	6	1	2	...	4	...	7	3	3	..	..	..	9	24	16	4	0	5	6	9		33	3.22
Mitral stenosis	15	17	3	2	5	8	2	4	1	...	4	2	...	1	..	...	...	...	20	12	10	2	0	3	16	4		32	3.13
Mitral stenosis and mitral incompetency.	13	19	4	7	4	3	2	...	...	3	1	4	1	2	1	...	..	...	19	13	13	4	1	2	9	6		32	3.13
Tricuspid incompetency	7	4	4	3	...	1	...	...	2	...	1	...	...	...	...	...	...	...	9	2	2	2	0	1	6	0		11	1.07
	612	412	41	32	69	95	159	71	80	46	110	66	113	73	39	28	1	1	400	624	383	100	91	184	218	324		1024	100.00

[1] Including 6 cases of chorea.

An analysis of the ages yields a number of interesting results. Up to the sixtieth year of life, the period at which valvular disease of the heart occurs least frequently is between the first and tenth years. After the sixtieth year there is a sudden decline in the number of cases, probably because many have already succumbed to the effects of the disease.

During the first decade of life the sexes show almost an equal predisposition to mitral disease, while during the second decade a considerable majority of cases occur in women. Statistics show that during this period of life acute rheumatism affects the female more frequently than the male, and this observation is confirmed by the fact that we find from the table, that at this age mitral disease occurred in 81 women and 52 men. In the decade between twenty and thirty, however, we enter upon that period of life most prone to endocarditis.[1] At this time, as men by their mode of life and occupations are more predisposed to acute rheumatism than women, and as the mitral valve, as already stated, is the one most frequently affected by rheumatism, the statistics under consideration show that 122 males were affected by mitral disease and only 58 females. In the succeeding two decades, also, the preponderance of instances of mitral disease is with the male rather than with the female, while subsequently the relative positions of the sexes are again reversed.

As would naturally be supposed, but a small proportion of the cases of aortic disease are shown to occur in early life. It is not until after the thirtieth year that the effect of those causes which eventuate in the production of sclerotic changes begins to be evident. Thus, prior to the thirtieth year we find that there were only 105 cases in which the existence of aortic disease was discovered, while after the thirtieth year the number of cases showing aortic disease was 287. Or if, in order to have for comparison two periods of equal duration, we deduct from the latter figure those cases occurring after the sixtieth year, the proportion will be found to be 105 cases from the first to

[1] Rosenthal: Real-Encyclopädie der gesammten Heilkunde, 1887, Bd. ix. S. 391.

the thirtieth year, and 253 cases from the thirty-first to the sixtieth year.

The cases of tricuspid disease noted number 11, the lesion consisting in incompetency of that valve. In every instance the disease was primary and not a relative incompetency depending upon mitral disease. In order that we may understand the reason for the non-occurrence of the latter condition in a single instance, it must be remembered that these statistics include but few cases that presented cardiac symptoms, and it seems hardly possible that mitral disease should progress so far as to produce relative tricuspid incompetency and at the same time cause no rational signs of cardiac disease.

A point of extreme interest in the analysis of these cases of tricuspid disease is the age at which the majority of them were found. In 7 of the 11 cases the age was between the first and the tenth year. In only one of these cases was a personal history of rheumatism obtainable; but in four of them, three males and one female, an hereditary tendency to rheumatism was clearly made out, in one instance upon the paternal, in the in the other three upon the maternal side. The presumption seems, therefore, justified that, in these cases at least, the lesion was congenital, the result of endocarditis occurring during fœtal life, at which time, as is well recognized, the right heart is more prone to suffer from endocarditis than the left.

A result shown by the table that I am unable to explain is the relative infrequency with which pure mitral stenosis, or mitral stenosis combined with incompetency, is found to occur. Guttmann,[1] in stating the relative frequency with which the various valve lesions are found, places the combined lesion of mitral stenosis and incompetency directly after the lesion of pure mitral incompetency. So also, Middleton's statistics, already quoted, show pure mitral stenosis and mitral stenosis combined with incompetency to occur in almost as many cases as do all the lesions of the aortic valves combined. In this connection, also, Sansom states that the most frequent combined

[1] Real-Encyclopädie der gesammten Heilkunde, 1887, Bd. ix, S. 353.

murmurs, restricting the word combined "to murmurs of differing rhythm generated at one orifice" are the presystolic and systolic mitral of stenosis and incompetency; and the table of F. J. Smith, quoted by Sansom, shows this lesion to have occurred in nearly 9 per cent. of the cases analyzed by him, occurring next but one in frequency to the lesion of mitral incompetency. The presystolic murmur of mitral stenosis, uncombined with the systolic murmur of mitral incompetency, is, however, shown by Dr. Smith's tables to have occurred in 3.38 per cent. of his cases—a result which corresponds very closely with that noted in the table upon which the present article is based, where it will be seen that this lesion occurred in 3.13 per cent. of the total number of cases.

Pure aortic stenosis, uncombined or unassociated with other valve lesions, is found to occur in a much larger proportion of cases than is shown by the table of F. J. Smith or the statistics adduced by Middleton. In explanation of this variance I must again call attention to the fact that but few of the cases here considered presented cardiac symptoms: hence complex lesions, which are so much more likely to produce these symptoms, are relatively few in number.

An analysis of the various factors in the antecedent history of the cases to which might be ascribed the causation of the valve lesions shows that a clear history of rheumatism, or rheumatic pains, was obtained in 383 cases, or 37.40 per cent. of the whole. An antecedent history of the infectious fevers was the only cause that could be adduced to account for the endocarditis in 100 cases, or nearly 10 per cent. of the total. These cases include only those in which one or more of the infectious fevers constituted the sole ascertainable cause to which the disease might be ascribed. In those instances in which a history of the infectious fevers was obtained, in conjunction with a history of the more probable causes of endocarditis, such as rheumatism, alcoholism, or syphilis, the latter were considered as the etiologic factors in the production of the disease.

A history of syphilis, undoubted or obscure, was obtained

in 91 cases, or nearly 9 per cent. of the total; and a history of alcoholism in 184 cases, or nearly 18 per cent. of the total. In all the cases in which a history of syphilis or of alcoholism was elicited, decided arterial sclerosis or atheroma was present; and in this connection it may be well to call attention to the large number of cases in which this condition of the blood-vessels was present. In 324 cases, or over 31 per cent. of the whole, disease of the vessels existed, and in many instances arterial sclerosis was the only apparent cause for the valvular lesion.

An interesting fact, to which I would call attention, is the large number of cases for which no cause could be assigned. These number 218, or over 21 per cent. of the whole. In Middleton's cases a history of rheumatism was obtained in over 62 per cent. of the cases, while in over 37 per cent. no rheumatic history could be elicited. That so many cases of chronic valvular disease of the heart should exist without evident cause is remarkable. Many of these cases were found in young individuals whose occupations entailed no overstrain, and from whom, by the closest questioning, no history of rheumatism, or even rheumatic pains, could be obtained. Upon close inquiry into the family history, however, the frequency of the occurrence of rheumatism in either parent was remarked. I think the knowledge to be gained from this fact is the probability of rheumatism producing endocarditis, even when the disease produces no evident disturbance of the functions of the joints; and that, therefore, the endocardium may, in some instances, be the only serous membrane that the rheumatic poison may attack. That this is a rational explanation of the occurrence of endocarditis in this class of cases is, I think, confirmed by the fact that in many of them a family history of rheumatism was obtained, that the age at which the disease was found was in many instances during that period of life most prone to rheumatism, and by the further fact that no other cause for the existence of the disease could be elicited.

In reference to occupation, it will be noticed that most of the cases occurred in those whose occupations were laborious.

The statistics were collated from among the working class of people, whose daily work, as a rule, entailed the severest kind of muscular exertion. Laborious occupations favor the development of cardiac valvular disease, for the most part by causing either heart-strain or arterial sclerosis. It is not surprising, therefore, that a history of hard work should have been obtained in such a large proportion of the cases.

In conclusion I would say that all doubtful cases have been excluded from these statistics; that, as far as possible, only cases of chronic valvular disease have been included, and that all cases possessing murmurs of possibly hæmic or functional origin have been carefully sifted out.

DISCUSSION.

Dr. Frederick P. Henry: One of the points that interested me in this report was the frequency with which valvular disease was found in the female sex. This, I think, was explained by the statement that the cases occurred among the working classes. As a general rule, taking the whole community at large, valvular disease is more prevalent among the male sex, and this is especially true of aortic disease. Among females of what are called the lower classes this does not hold good to the same extent, for they are exposed to the same privations and perform the same kinds of manual labor in many cases as the male sex.

With reference to hæmic murmurs, it is well known that such murmurs may be due to mitral regurgitation. It is not always possible to distinguish between a hæmic murmur so produced and one due to actual valvular disease. The term dynamic has been used to designate a mitral regurgitant murmur produced by a functional cause, the term hæmic being applied to anæmic murmurs at the base. It has been demonstrated that during systole the space enclosed by the mitral ring is reduced one-half. In anæmia when the cardiac muscle, like the other muscles of the body, is weak, this closure of the ring is imperfect and regurgitation results. Anæmia is not always rightly diagnosticated by external appearances. In the presence of a soft murmur, mitral regurgitant in character, without a history of rheumatism, syphilis, or alcoholism, or without any notable impairment of health, we would be led to suspect that the murmur was of blood origin. The only way to settle the point would be by noting the effect of treatment. If, under the use of hæmatinics, the murmur disappears, it is anæmic. If the anæmia persists

in spite of treatment, the exact character of the murmur may never be cleared up until the post-mortem examination is held.

The question of the frequency of aortic disease with pulmonary phthisis is one in which I have long been interested. I have found that the association of aortic murmurs with phthisis is not so rare. Phthisis with mitral regurgitation, or any form of lesion which flushes the lungs with blood, is very rare, for a lung flushed with arterial blood does not offer a suitable nidus for the tubercle bacilli. The reason that phthisis is not uncommonly associated with aortic disease is because aortic lesions are often the result of atheroma, and there certainly is no antagonism between atheroma and phthisis, in fact quite the reverse. There is, in my experience, a predisposition on the part of phthisical persons to have atheroma, and conversely, there is a predisposition on the part of a person with atheromatous vessels to develop pulmonary phthisis.

Another point shown by this paper is, that valvular heart disease may coexist with a fair state of the general health. This was first demonstrated conclusively by Sir Andrew Clark when he referred to about seven hundred cases of valvular heart disease existing for five years in individuals who had shown no deterioration of health which could be attributed to the cardiac affection.

Dr. Morris J. Lewis: I would ask if Dr. Ashton was able to elicit any history of antecedent chorea in these cases? It has been found at the Orthopedic Hospital and Infirmary for Nervous Diseases that the majority of cases of chorea show cardiac symptoms. Dr. Osler sometime ago took the trouble to look up the old cases of chorea, and found that a large proportion of them still had an organic murmur, and this independently of any history of rheumatism. He thought that chorea caused more cardiac disease than did rheumatism itself.

Dr. Ashton: There were only six cases of chorea included in this series, and in all of these there was also a history of rheumatism.

Dr. H. A. Hare: It is difficult to discuss a statistical paper because the statistician knows so much more about the subject than anyone else who has not gone over the figures. There are one or two points which occur to me. The first is the frequency with which murmurs occur as the result of a rheumatic taint, without the manifestation of the external signs of rheumatism in the joints. I heard Douglass Powell, in 1888, in his Brompton Hospital lectures, emphasize this point very strongly. He showed a number of cases where careless cross-examination failed to reveal any rheumatic history, but in which a more careful inquiry developed a distinct rheumatic taint. This could often only be traced by heredity, but in many cases by the occurrence of "growing pains." I then learned the importance of discovering these subacute histories of rheumatism in attempting to explain certain cardiac murmurs.

Another point of some interest, as showing the way in which statistics taken in different countries could vary, is the different conditions under

which people live and the different tendencies which are manifested in their systems. Any of us who have done clinical work in England must have been impressed with the fact that rheumatism is much more prevalent than in this country. In the Brompton Hospital it seemed that almost every patient with cardiac disease gave a history of rheumatism. Here we see almost no cases of rheumatism as compared with the number to be seen in England.

Another point is as to the desirability of accepting statistics which are based on physical examination, but which are not confirmed by autopsy. There are many murmurs which we hear, and which later entirely pass away. I thoroughly appreciate the fact that it is impossible to eliminate this fallacy It is only by comparing such statistics as these with those made at the post-mortem table that we shall be able to come to any certain conclusions. I recall the case of a girl, aged sixteen years, who evidently had an aneurism of the aorta with aortic valve disease. Careful watching showed that the aneurism was extending and apparently involving the innominate. This patient was sent to one of our colleges and lectured on as an unusually interesting case of aneurism of the aorta and innominate. She was lectured on three times. At the third time the lecturer, who was lecturing on her for the second time, said he believed that it was a case of hysterical aneurism. The girl died, and at the autopsy, which was made by Drs. Osler, Griffith, and myself, nothing was found the matter with the valve, and there was no aneurism of the aorta. The vessel was, however, extraordinarily distensible.

With regard to phthisis and valvular lesions, mentioned by Dr. Henry, if phthisis occurs so rarely where the lung is flushed with arterial blood, we should expect it to frequently occur in emphysema, where the lung is not flushed with arterial blood. While we do have phthisis associated with emphysema, it is by no means as frequent as we should expect, reasoning from the analogy.

Dr. James B. Walker: Reference has been made by the author to the absence of a history of rheumatism in many of his cases. In a recent report of a lecture by one of our clinical lecturers, in speaking of a case of valvular disease where no history of rheumatism could be elicited, it was said that no doubt rheumatism had existed, as the lesion of the valves was there. I was glad to hear that such carefully prepared statistics could show so many cases where there was no history of rheumatism. There is no reason why there should be. I am satisfied that in a great many cases there is no personal history of rheumatism, although there certainly is in most instances a history of rheumatism in the family. Valvular heart lesions are not hereditary, but the causes that lead to them are, and the various rheumatic and gouty phases are here prominent. As a case in point: I know a mother and three daughters; the mother had a mitral valve lesion with a distinct history of rheumatism. She had in her previous life been a farmer's wife and exposed to greater or less hardships; her daughters had not been exposed to such influences; none of them had rheumatism, but all three have mitral lesions,

and one a double mitral lesion. I have seen similar inheritances in many cases. There is an evident tendency to the inheritance of conditions favoring valvulitis. In the vast majority of cases I have no doubt that it is a hæmic condition, not sufficient to induce arthritic trouble, but enough to induce endarteritis and valvulitis. I am certain that rheumatism need not exist in such form as to give rise to arthritic trouble in many cases of valvulitis any more than that gout need be present as podagra in order to establish gout as the potential factor in the induction of endarteritis.

As to prognosis, there is no question that a young patient can recover from a valvular lesion even when associated with endocarditis of rheumatic origin. Several years ago I attended a girl, ten years of age, with endocarditis associated with inflammatory rheumatism. She was also choreaic, and for a long time exceedingly anæmic. She grew up, married, and became pregnant. She still had the cardic lesion, although the health was somewhat improved. There was no trouble at the labor, and she has since had two children without trouble, her general health improving with age, though the lesion still exists. I am in the habit of giving a more favorable prognosis in heart disease in young adolescents than in older subjects. There is in young persons a capacity for compensatory hypertrophy and conditions which favor a return to fair health, even though the lesion still remains.

Dr. Charles W. Dulles: I would like to ask Dr. Ashton what was the total number of persons he examined in preparing his tables? The value of his statistics would be better understood if he were to state the number of persons that he examined as well as the number of cases of organic disease of the heart that he discovered, so that others may know the proportion of the latter to the former.

Dr. Henry: I do not think that it can be too strongly emphasized that there are other manifestations of rheumatism besides the articular symptoms. This point was discussed two years ago at the meeting of the Association of American Physicians, in connection with the paper of Dr. Morris J. Lewis on "The Seasonal Relations of Chorea." We recognize rheumatic tonsillitis, rheumatic pleurisy, and various rheumatic eruptions, especially erythema nodosum. These points must be borne in mind when questioning the patient with reference to a previous history of rheumatism.

Dr. Hare: There are two other points to which I might refer. One is the importance of not confusing those cases in which compensatory hypertrophy has occurred with those in which the murmur disappears, and the other is the frequency with which loud murmurs due to endocarditis will pass away, particularly in young subjects. I recall the case of a young married woman, aged nineteen years. She had an exceedingly severe attack of acute rheumatism during which she aborted. A murmur developed and persisted so long that I gave an unfavorable prognosis. She, however, not only got entirely well, but skilful examiners reported that she had no murmur, and I had myself an opportunity of confirming this observation.

It is generally customary among many clinicians to teach that mitral regurgitant disease is the most frequent, next aortic obstructive, then aortic regurgitant, and then mitral obstructive. I should like to ask Dr. Da Costa if this is his general impression. Some years ago a teacher of clinical diagnosis in this city in one winter seemed to have an epidemic of mitral stenosis. Nearly every case was said to present this lesion. Others, however, could not detect these murmurs.

Dr. J. M. Da Costa: My experience is in the general direction indicated by Dr. Hare. I think that true mitral stenosis is rare, though not the rarest of valvular heart diseases. Diseases of the valves of the pulmonary artery are the rarest, especially constriction. Tricuspid affections are far less frequent than those of the mitral or the aortic valves. I am astonished at the number of cases of tricuspid insufficiency referred to in the paper.

I understand that these examination include as many women—if not more—as men. This is an important point to be borne in mind, as life insurance records are usually those of men.

Dr. A. A. Eshner: There is a point in these statistics concerning which there is a possibility of fallacy, and that is the question of age. As the lesions were discovered incidentally in the course of routine, systematic examination, the period of life at which the cases came under observation bears no relation to the time of the occurrence of the lesion.

Dr. Ashton has referred to the comparative infrequency of mitral obstructive lesions in his statistics. The explanation, I think, lies in the fact that, as he states, the cases came under observation in a condition of presumed health, while obstructive interference at the left auriculo-ventricular orifice would have been likely to give rise to subjective manifestations.

Another interesting point is that of etiology. I am myself impressed with the fact that we lay too much stress upon the importance of rheumatism as a cause of endocarditis. It often happens that if in any given case of valvular disease a history of rheumatism is not obtained, the case is put aside as one of faulty observation. Undue importance has been attached to vague pains and anomalous symptoms as indicative of a rheumatic condition. If modern investigation has shown anything it is that inflammation of the serous membranes may result from a host of conditions. It may result from poisoning by lead, or by alcohol, or as the result of syphilis. We have learned that in addition to rheumatism, it may be a complication of chorea, of gonorrhœa, of influenza, of puerperal infection—in fact there is not a single infectious or toxic condition that may not give rise to endocarditis. This fact has not received the consideration it deserves, as the tendency has been, in the absence of a direct history of rheumatism, to infer that the attack had been overlooked, or to ascribe some vague pain or symptom to a rheumatic origin.

TREATMENT OF TYPHOID FEVER.

By CHARLES W. DULLES, M.D.

[Read November 7, 1894.]

The recent appearance of articles on the treatment of typhoid fever, advocating principles similar to those that have impressed themselves upon me in the course of my practice, prompts me to present the views on this subject that I have come to hold after nearly twenty years of observation and experience. Though these impressions differ in some ways from those that are strongly urged by certain writers on typhoid fever, I present them with confidence, because I see in various places that opinions I once held with hesitation, as unorthodox and somewhat hazardous, are held by others, independently drawing conclusions from their own experience.

My experience in the treatment of typhoid fever has ranged from cases so mild as to leave some doubt in regard to the diagnosis to cases so severe as to be marked by hemorrhages from the bowels, kidneys, and lungs, with delirium and collapse; so that I have had sufficient opportunity to test the teachings of others and the deductions from my own observations since my student days.

The principles of treatment of typhoid fever may be summarized, I think, as consisting in: first, careful maintenance of the natural processes of excretion; second, the judicious administration of food—finding a mean between starvation and overloading the alimentary canal; third, moderate regulation of temperature; and, fourth, medication reduced to the lowest possible point and shaped to meet definite indications.

Nothing in these principles is novel; but I do not think the text-books put them as forcibly as might be done with advantage to students and young practitioners.

Concerning the first matter, I would say that the views in regard to auto-intoxication, expressed by Bouchard at the International Medical Congress held at Geneva, in 1877, impressed me at that time as being of very wide applicability. He applied them especially to typhoid fever, in which disease, if ever, we have an accumulation in the intestinal canal of material which taxes to the utmost the powers of elimination possessed by the patient. Here, if ever, we need to see that no interference with the process of excretion takes place, but that, on the other hand, aid shall be given to the excretory function of the lungs, the skin, the kidneys, and the bowels. For the lungs, we need fresh air, with careful avoidance of undue chilling; for the skin, we need, again, abundance of fresh air, judicious management of bed and body coverings, and the application to the skin of some form of hydrotherapy. To the kidneys unremitting attention should be given in every case of typhoid fever. The urine should be examined often for albumin and for tube-casts; and in every case the quantity of liquid drunk by the patient and the amount of urine passed should be known. I believe that much of the delirium of typhoid fever might be avoided if the kidneys were considered as carefully as the intestines. During the entire progress of a bad case the tendency of the urine to hyperacidity should be combated by the free use of water and dilute alkaline solutions. From my experience I should say that the best form of the latter is the solution of ammonium acetate with the addition of potassium acetate.[1] I have found it desirable,

[1] To make a preparation of ammonium acetate and potassium acetate that is not very disagreeable, I order a mixture containing one hundred and sixty grains of potassium acetate in three ounces of the solution of ammonium acetate sweetened with one ounce of syrup of lemon. To this I add ten grains of quinine sulphate, and order the mixture to be filtered by the druggist. The dose is a dessertspoonful, taken with a wineglassful of water, about four times a day. The quinine substitutes for the mawkish taste of the potassium a distinctly bitter taste, and that part of it (which is considerable) that is not dissolved in the mixture is filtered out, in order to make a better-looking preparation. To each dose I sometimes recommend the addition of a few drops of lemon-juice.

also, in these cases to induce patients to drink water freely by giving it to them in forms which are agreeable to sick persons as well as to well ones; for example, carbonated waters, or plain water slightly acidulated with lemon-juice, or weak tea, hot or cold, flavored with a little lemon-juice and sugar, in the manner so widely known as "iced tea."

Of the various modes of elimination, I have chosen to refer last to that from the bowels, because it is in some respects the most important and in some respects the most neglected. My own observation leads me to think that too great fear is sometimes felt for the diarrhœa of typhoid fever. This diarrhœa is the result of Nature's efforts to get rid of material that is injurious to the patient: and I believe, on the one hand, it would be less likely to occur if the physician secured proper evacuations from the bowels before the diarrhœa began, and, on the other hand, that lives are sometimes sacrificed in a desire to check it. This endeavor seems to overlook the indications furnished by the natural course of a case of typhoid fever, as well as the principle that the contents of foul cavities in the body—whether these be natural cavities, like the pleural cavity or the peritoneal cavity, or unnatural cavities, like those of abscesses—shall be removed. I think it important at the outset of a case of typhoid fever to secure a thorough clearing out of the entire intestinal canal—not merely a single purging, but several moderate ones, which shall quite empty the whole tract. After this I believe that the bowels should be relieved with mild saline laxatives, like Rochelle salts, or with small and repeated doses of calomel, at least every third day, unless the case is one in which Nature provides this form of relief. As for astringents, I think the occasions for their use are extremely few. This is especially true of astringents administered by the mouth. Astringents administered by rectal intubation, I think, are of little more value than simple lavements of the sort now called "enteroclysis," but which the students of Monti became familiar with some twenty years ago in Vienna, where it was called "irrigation of the colon." This I have practised on suitable occasions for seven-

teen years. Such irrigations are useful in several ways—for cleansing, for cooling, for assuaging thirst—but at this time I do not care to discuss details. The sum of what I believe in regard to elimination from the bowels is that it should be facilitated, and that, instead of attempting to check it, the object should be to regulate what Nature may be doing in an irritating and irregular fashion.

The second important point in the treatment of typhoid fever is, I think, the judicious administration of food. It goes naturally with what has been said in regard to checking Nature's efforts at elimination, to say that Nature should not be harassed by having burdens laid upon a weakened and irritable intestinal canal. The teachings of physiology and of ordinary common sense are opposed to the introduction into the alimentary canal of a sick person of more food than he can either appropriate or cast out. A healthy person can dispose of a large excess of unneeded ingesta, but a patient with typhoid fever cannot. I am sure I have seen patients killed and others nearly killed by relentless feeding, practised by physicians who have, without judgment, followed the teachings of writers who advocate what is called "regular" feeding, and especially with milk-food, in typhoid fever. Forcing patients to take measured quantities of milk at short intervals sometimes results in a diarrhœa which shows that Nature repudiates the imposition—the stools resembling those of a sick, milk-fed infant. At other times, when milk is given, the milk-water and salts are absorbed while the casein remains undigested and accumulates in the lower bowel until a condition of coprostasis is set up, in which the patient is subjected to the dangers of total obstruction to elimination from the bowels and to horrible pains and violence when the mass of solid feces is finally expelled by an act of Nature or by the stimulation of cathartics.

An error of another sort is committed when certain extracts of beef are administered with the idea that they are highly nutritious. To support this belief there is, I think, only an untenable theory and deductions which the facts do not war-

rant. I feel sure that the advantage to patients of preparations such as Valentine's meat-juice is chiefly that they are so costly that not much is likely to be given, and that they contain so little tissue-building material that they do not oppress the digestion or overload the intestinal canal. Such food probably contains, bulk for bulk, less tissue-building material than is found in the white of chicken's egg, and is equally innocent. Beef-tea is now generally recognized as a substance which is useful for the same reason, namely, that it does not burden the intestinal canal, while it gives both patient and friends a sense of security which is very desirable in all medical cases.

This suggests what I believe is one of the most important points in the management of typhoid-fever cases, namely, that they should be given as little food as possible—not as much as can be forced down them. My own rule—which I have found has been that of others—in almost all cases of sickness, in young and old, is to say that patients may eat when they wish to, and go without eating when they have no desire for food. I let them drink all they want to, and give them food which shall resemble as little as possible those trying things that remind a patient of his sickness. I give a list of permissible foods, and tell the nurse or members of the family to get up a little meal for a patient who is conscious enough to appreciate it. I sanction the use of the albumin-water already referred to (cool water in which the white of an egg has been stirred up, with the addition of some pleasant flavor), and of almost all clear soups as they are prepared for well persons. I give milk, plain or peptonized, or made more palatable and less constipating by the addition to it of some good preparation of cocoa. I allow tea and coffee, with plenty of cream and milk, which often do good, and which I have never known to do harm if used with ordinary judgment. I have no fear of eggs, and give them whenever a patient with typhoid fever or any other fever wishes them, either soft-boiled or shirred.

Besides this, if I have a patient whose bowels are acting nicely I give custards and simple corn-starch preparations. A patient who can digest anything can digest these foods;

and a patient who cannot digest anything will not want anything to eat; and, according to my views, he will require nothing but albumin-water and thin soups.

When we recall, on the one hand, the fact that persons making more or less constant exertions have gone for weeks without food and without serious impairment to their health, and, on the other hand, that fevered intestines are in no condition to do much in the way of digestion, and are capable of taking up only foods which contain finely divided or soluble hydrocarbonaceous substances and albuminoids that make their way through the intestinal walls without digestion, we can, I think, understand that not much food is needed to keep fever-patients from starving, and that this food should be of the very simplest character.

Further, when we reflect that perforation of the bowels is most likely to occur when the bowels are obstructed and distended with gas, and that this accident does not result from the unimpeded movement of soft fecal matter, but that the intestines are rather advantaged by the internal drainage which such a process secures, we can understand that, while the bowels are not constipated but moving freely, there is no need to restrict a patient to food like milk, which is almost entirely composed of water, and which has but little residue. Because I think this is true, I believe that typhoid-fever patients who have reached the stage in which they feel an inclination for food may be allowed moderate quantities of any soft food, like thick soups (digestible, of course), good fresh fish, and the soft part of oysters, with occasionally sweetbread or beef's tongue, with eggs, milk and cream. Of vegetables, I think baked or mashed potatoes may be used, and rice and corn-starch preparations, with thoroughly boiled oatmeal and such things as spinach or well-boiled onions and very moderate quantities of toasted bread thoroughly softened with boiling hot water and seasoned, as well as soft milk-toast. For relishes, cocoa, tea and coffee may be used, and, for desserts, jellies, ice-cream, and orange-juice in moderate quantities.

The third point in regard to the treatment of typhoid fever

about which I wish to speak is the regulation of the temperature. Views on this subject have undergone considerable modifications within the time covered by my own experience. At one time the greatest stress was laid upon lowering the temperature of fever-patients. This plan has given way to a more rational one, that recognizes a principle known as long ago as the time of Asclepiades, two thousand years ago—namely, that fever may of itself be a curative process or the mark of a process which is useful. I think the majority of thoughtful medical men are entirely satisfied if a patient presents the general appearance of doing well, even though the temperature be several degrees above 100° Fahrenheit. If a patient is really suffering from high temperature I think the first attempt to reduce it should consist in having removed the needless bed-covers with which fever patients are often burdened, as the air constitutes one of the best and safest means of properly reducing the temperature. After this, I have found that sponging with cool water, especially upon the abdomen, is pleasant to the patient, and I think it has a distinctly beneficial influence upon the temperature and the inflammation within the abdominal cavity. In some cases I advise that a few light layers of muslin or gauze such as surgeons use be laid upon the abdomen and moistened at times with ice-water gently squeezed from cotton or a sponge. In a few cases I have found it convenient to have such light cloths "ironed" occasionally with a small piece of ice.

My experience with drugs given to reduce temperature by means of their physiological action has led me to abandon their use; not because I have ever seen them do harm, but because I do not believe they really do good, and I think it better to let the temperature alone as long as it is in bounds, and, if it requires repressing, to effect this with external applications of cool air and cool water. Since I have adopted this plan I have found it to work according to the expression attributed to Asclepiades, namely, "*cito, tuto et jucunde.*"

This suggests the last of the points which I have mentioned as constituting, in my opinion, those most important in the

treatment of typhoid fever, namely, that which concerns medication. I am not peculiar in believing that medication should be reduced to the lowest point and that it should be shaped to meet definite indications. As I have already said, I think the bowels should be well opened at the beginning of a case believed to be one of typhoid fever, and that when the diagnosis is established they should be kept open. I have also suggested the use of mild diuretics, the tendency of which is to reduce the acidity of the urine and to increase the solubility of its solid contents. With these procedures, intended to aid elimination, I think it is useful to administer what are known as intestinal antiseptics. These appear to do something which is not yet explained by any laboratory observations; and, from the time when turpentine was first used until the present time, when it is the rival of modern chemical inventions, intestinal antiseptics have seemed to be of distinct value in the treatment of typhoid fever; for in addition to their purifying action in the bowel, they appear to exercise a beneficial influence upon the nervous system. Turpentine and its congeners have tonic and stimulating properties in addition to their antiseptic influence, and drugs like salol probably control pain, not only by acting upon the fluids circulating in the blood-vessels and lymphatics, but also by directly influencing the brain and nervous system. I think turpentine and salol are two valuable drugs, if given in moderate quantities and with care that the turpentine shall not be pushed to the point of producing strangury, and that the salol shall not be pushed to the point of producing carbolic-acid intoxication. In certain conditions I think morphine may be used, and should be used, without hesitation. But these circumstances are rare. The bromides (and I prefer sodium bromide) are useful as occasional remedies. So is sulphonal, and so is trional, to control restlessness and to secure sleep, in the absence of pain, on which they have no effect whatever. Phenacetine is also useful with a view to controlling nervous irritability, bearing in mind its effect in producing perspiration. I believe that at times pepsin-solutions, with dilute hydrochloric acid added, are use-

ful as an aid to digestion and the natural processes in the intestines, and not as medicine.

My view in regard to alcoholic stimulants is that they are seldom required, and never in the large quantities sometimes prescribed. There are times when a dose of a teaspoonful of whiskey in a little hot water, repeated every ten minutes, may tide over a short period of weakness or collapse; but I think the administration of such quantities as eight or sixteen fluid-ounces of whiskey in twenty-four hours is not good practice; while I believe that hot tea or coffee, without cream or milk, may be used instead of the smaller quantities of alcohol just mentioned.

I have not attempted to describe in detail the mode of treating typhoid fever, but only to state the general principles which impress themselves upon me as correct after a reasonable experience. These are, as stated: 1, careful maintenance of the natural processes of excretion; 2, the judicious administration of food; 3, moderate regulation of temperature; 4, medication reduced to the lowest possible point. My object in discussing the subject at this time is not so much to present my own opinions as to put them in evidence in order to strengthen the hands of those who have recently contended for what I believe is the rational mode of treating this very serious disease.

FURTHER OBSERVATIONS UPON THE ETIOLOGY, DIAGNOSIS, AND TREATMENT OF ACUTE AND CHRONIC APPENDICITIS; WITH THE REPORT OF SIXTY-ONE CHRONIC CASES OPERATED UPON, WITH ONE DEATH.

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[Read November, 7, 1894.]

Since the publication of my last papers on appendicitis, which appeared in *The Medical News* of May 19, May 26, and June 16, my further experience has more than convinced me that the views then expressed have been borne out by the results attained by the measures then advocated. Further, I am prepared to express myself more strongly than ever in favor of early operation in acute primary attacks of appendicitis, and of operation in all cases of chronic appendicitis, including under this heading the sub-acute, the relapsing, and the recurrent varieties.

As regards etiology, I would emphasize the views I have already expressed regarding the importance of foreign bodies as a factor in the causation of a large proportion of acute and of a smaller number of chronic cases. In the great majority of the latter the condition found is that of a chronic catarrhal inflammation, while the bulk of acute cases are due largely to foreign bodies, *i. e.*, fecal concretions and extraneous substances. I have only seen two cases in which real foreign bodies were not found. In one, an acute case, the appendix contained a

large number of strawberry seeds; in the other, a chronic case, the appendix had contained a date-seed which had escaped at the point of ulceration. It was found above the tip of the appendix, which pointed north and lay behind the colon.

I believe that all cases of appendicitis begin as a catarrhal inflammation, in which the bacterium coli commune plays an important causative rôle. After this inflamed condition of the appendix has been established, the future outcome of the case, pathologically, very frequently depends upon the presence of a fecal concretion or foreign bodies and bacteria of suppuration other than the bacterium coli commune. No definite rule can be laid down, because there are cases that illustrate both conditions. It has been my experience that in acute perforative, and frequently in non-perforative, cases fecal concretions were found to exist either within the organ or in its immediate neighborhood.

I have already made the statement that the diagnosis of appendicitis is not difficult in the vast majority of cases. I am now more than ever convinced of the truth of this statement and of the importance it bears to successful treatment. The history of the case and the localized signs that centre around that most valuable landmark known as McBurney's point are always sufficient to establish the diagnosis either directly or by exclusion. The acute cases that go on to suppuration, and in which there is the greatest tendency for the pus to become circumscribed, are those in which the appendix points toward the northern end of the appendiceal compass and lies between the layers of the mesocolon.

The palpation of the appendix in chronic cases is a valuable and reliable means of diagnosis. Of course, in those cases in which the organ lies behind the cæcum the method is less valuable, although it is even then of service, because when the cæcum is distended the condition is due to flatus and not to feces. This has been my experience in sixty-one chronic cases upon which I have operated, to say nothing of a much greater number of acute cases. I have been able to diagnosticate and demonstrate by operation a thickened appendix, giving its

direction and location and its depth from the anterior wall of the abdomen. Women are better subjects, but the method is applicable to men, especially if they have been the victims of repeated attacks of appendicitis. In chronic cases I have noted that upon palpation over the base of the appendix the pain is referred in a direction corresponding to the long axis of the organ, *i. e.*, when the pain is referred to the liver the appendix points north, and so on, corresponding to the different positions that the appendix holds.

The difficulty attending the differential diagnosis between chronic appendicitis and incipient psoas-abscess, that is before the pus has passed any distance down the psoas-sheath, I have had forcibly brought to my mind recently in two cases. The chief points in favor of a forming psoas-abscess are the appearance of the patient, usually that suggestive of tuberculosis, the information to be obtained by an examination of the spine, a complete temperature-record, and a tendency to flexion of the thigh of the affected side. While the last-mentioned sign may be and is present in some cases of chronic appendicitis, it is a far more frequent accompaniment of psoas-abscess. Palpation will, in the great bulk of cases of chronic appendicitis determine the presence of a diseased appendix, while deep pressure over the right iliac fossa will in case of psoas-abscess, although revealing tenderness, fail to disclose the presence of either a diseased appendix or the characteristic rigidity of the flat muscles of the abdominal walls.

As regards treatment, my rule is this: Given the case of a patient who suddenly develops severe abdominal pain, at first referred either to the epigastrium or about the umbilicus, and later to the right iliac fossa, accompanied by nausea and vomiting, and following some dietary excess or indiscretion, and preceded as a rule by a history of intestinal dyspepsia, and when examination reveals rigidity and tenderness over McBurney's point, I at once administer a purgative, preferably castor-oil or salts. If this is not retained I give calomel. If after the purgative has operated satisfactorily the symptoms continue, along with an increase of intensity of the localized tenderness,

I at once operate. The three cases that follow will illustrate my statement and prove my theory to be sound, and the procedure a life-saving measure.

Case I.—On the evening of June 26, 1894, I was called to see Dr. J. H. B., aged forty-five, one of our leading practitioners. I found him suffering with acute abdominal pain, most intense in the right iliac fossa, the character of which I recognized as that of appendicular colic. He gave a history of chronic intestinal dyspepsia extending back for a period of years, also a vague history of having suffered in the past from a slight attack of appendicitis. Three days prior to my visit he had suffered from an acute attack of indigestion, the result of indiscretion in diet, which had been somewhat relieved by active purgation. The day of my visit he had had several bowel-movements, yet the pain in the right iliac fossa, which was paroxysmal, was increasing in intensity, notwithstanding the fact that he had taken, upon his own responsibility, one-fourth of a grain of morphine. Physical examination of the abdomen convinced me that the man was suffering from an acute, progressive attack of appendicitis. I advised immediate operation, and at 8 o'clock the following morning I removed the acutely inflamed appendix, the mesenteric attachment of which was gangrenous in its distal half. Recovery was uninterrupted.

Case II.—Dr. J. C. R., aged twenty-two years, resident physician in the German Hospital, while on duty was taken sick on the evening of August 30, 1894, with severe general abdominal pain, soon becoming localized in the right iliac fossa. Under active purgation he was somewhat relieved, but the appendicular pain persisted. On the evening of the 31st I was asked to see the patient by Dr. Frese, the chief resident physician of the hospital, who informed me that in his judgment the doctor was not so well, and he feared the case was progressing unfavorably. I confirmed Dr. Frese's diagnosis of acute appendicitis and advised immediate operation.

The operation was done at 8 P.M. The appendix occupied the southwest position and was bound down by adhesions. It was gangrenous and about to perforate; it contained a small collection of pus at its tip. Recovery was uninterrupted.

Case III.—During the early part of the past summer I was called to see Mrs. B., the wife of a prominent Philadelphia physician, who was suffering from what was believed to be appendicitis. She was at the same time suffering from uterine hemorrhage, which from its severity and from the fact that it was associated with acute paroxysmal abdominal pain, raised the question of extra-uterine pregnancy. Vaginal examination demonstrated a point of excessive tenderness high up and to the right, but otherwise was negative. I confirmed the diagnosis of appendicitis, and we decided upon active purgation. This not being followed by abatement of her symptoms, operation was advised. The appendix lay southwest, was adherent, very much enlarged, and con-

tained a considerable amount of pus. The surrounding tissues were covered with inflammatory lymph. Recovery was uninterrupted.

In this connection it is interesting to note that this was the third member of the family upon whom I had operated for appendicitis.

Case IV.—I recently saw in consultation a young lady with appendicitis in whom the symptoms had not yielded to purgation; the appendix was so palpable that it was believed to lie in contact with the anterior parietal peritoneum. Operation was advised. The appendix was found acutely inflamed and in contact with the anterior parietal peritoneum. Recovery was uninterrupted.

I report the following two cases of acute appendicitis to illustrate the frightful rapidity with which such cases may go from bad to worse if not operated upon very early. The only possible hope of recovery in such cases lies in immediate operation. These are the cases that do not show decided improvement with marked amelioration of all symptoms, especially of tenderness, after the administration of purgatives.

Case I.—Mr. R., aged twenty-eight years, was attacked fifty hours before operation. The symptoms grew steadily worse in spite of all medication. When the belly-cavity was opened pus welled up in the wound in quantities. The appendix, which pointed northeast, was gangrenous and had separated from its attachment to the cæcum, which also was gangrenous, leaving a large hole in the latter, through which fecal matter was escaping. The opening in the cæcum was closed with difficulty. The peritoneal cavity was carefully and thoroughly washed out and drained with glass tube and gauze. The patient died on the third day following the operation.

At the post-mortem examination the external wound was found in good condition, and the glass and gauze-drainage still in position. The omentum was congested and infiltrated, presenting the appearance of a cock's comb, and was adherent to the lower end of the cæcum around the drainage-tube. All the tissues in the right iliac fossa were in a semi gangrenous state. The cæcum around the opening found at the operation was gangrenous; the stitches, however, were still in position. To the inner side of the row of sutures the bowel was perforated, allowing the escape of feces and pus. The general peritoneal cavity was infected, but contained very little pus; there was no pus in the pelvis. The intestines were covered with lymph. The cause of death was septic peritonitis.

Case II.—Mr. K., aged twenty-three, was attacked May 23 with severe abdominal pain, referred to the epigastrium, and soon becoming localized in the right iliac fossa. Tenderness was marked and persistent, and on the 25th was intense, accompanied by exaggeration of all the symptoms, local

distention, vomiting, and constipation. Operation was undertaken on May 28th. When the peritoneum was opened a considerable quantity of pus escaped. The cæcum was distended with gas, and the small intestines were injected, but not paralyzed or distended. The appendix, which occupied the northeast position, was brought into view and tied off. The meso-appendix was short, and was attached to the basal half of the organ; perforation had taken place at about the middle third; the appendix beyond was gangrenous. There were no adhesions, and apparently no attempt upon the part of nature to close off the general peritoneal cavity. The pus cavity was thoroughly washed out, but upon placing glass-drainage in the pelvis fully a pint more of pus escaped. The patient made a rapid and safe recovery.

The amount of pus, especially that in the pelvis, and the absence of any apparent attempt upon the part of nature to protect the general peritoneal cavity, and the recovery of the patient, point conclusively to the fact that with proper technique the general peritoneal cavity under these circumstances can be protected against infection, and the case brought to a successful issue.

In connection with this paper I report 61 cases of operation for chronic appendicitis, with one death. The fatal case was the following:

B. K., a female, aged twenty-two, born in Ireland, was admitted to St. Agnes' Hospital, September 19, 1894, with a history of four previous attacks of appendicitis. At the time of admission to the hospital she complained of pain in the right iliac fossa. The tenderness in this region was so great upon slight pressure as to preclude a thorough examination. The greatest tenderness was at the McBurney point. Immediately beneath the right semi-lunar line and within the abdomen a large mass was felt. The patient suffered from retention of urine, requiring catheterization.

Upon opening the peritoneal cavity an immense mass came into view, composed of the small and large intestine and omentum, bound firmly together by dense adhesions. The omentum was ligated in sections and cut away. The adhesions were carefully broken up, exposing the cæcum imbedded with the appendix in an inflammatory mass. After a tedious dissection the cæcum and appendix were freed, and the latter ligated and removed.

I beg to call attention particularly to this case on account, first, of the number of attacks; second, the condition found at the time of operation; and, third, the result. Had the

patient been operated upon early in or after the primary attack, the result would doubtless have been different. If the appendix had been removed at this time the inflammatory mass found in the right iliac fossa would not have been present, and such an extensive dissection not rendered necessary.

The rôle the appendix plays in many cases of intestinal indigestion I am sure is important. This has been beautifully shown in many of the cases of chronic appendicitis I have operated upon for digestive symptoms, which were so persistent before the appendix was removed, and entirely disappeared after recovery from the operation. I am so thoroughly convinced of the importrnce of this fact, that I believe a permanent cure can only be obtained in a certain proportion of cases of chronic intestinal catarrh by removal of the appendix, should it reveal tenderness, even when there has been no clear history of an attack of inflammation of this organ.

In my record of operations for chronic appendicitis are included three cases in which was present chronic diarrhœa with mucous stools, and in one also blood. In two of these cases the diarrhœa, along with other evidences of intestinal disturbance, disappeared six months after operation. In the third case, operated upon five months ago, while there is still diarrhœa, it is improving. This case is of special significance from the fact that, although the patient had received most exhaustive and prolonged treatment, both internal and local, at the hands of expert medical men, yet the diarrhœa proved rebellious. I believe this case will, as have the others, entirely recover.

In closing, I wish to emphasize the important deductions that have forced themselves upon me. The first and most important of all is the necessity of early operation for those cases of acute appendicitis, whether in the initial attack or in an acute attack supervening upon a chronic appendicitis not immediately yielding to judicious purgation. The ravages of this affection are so rapid and so fatal that I can hardly express myself too strongly upon this point. I hear so often from medical men and the more conservative surgeons that appendicitis is amen-

able to medicinal measures, yet when they call in the consulting surgeon for those of their cases which do not improve, how often are they found beyond surgical aid? How often are we called in at the last moment to see supposed cases of obstruction of the bowels or idiopathic (?) peritonitis, only to find the patient moribund, with cold, blue extremities, in fact profoundly septic, the victim of a perforative appendicitis! I could cite instance after instance in which patients have died of inflammation of bowels, peritonitis, obstruction of the bowels, and in one instance of "heart-failure," only to discover at the autopsy a gangrenous and perforated appendix with a belly full of stinking pus. In looking over the weekly mortality-reports of the Philadelphia Board of Health I have often been struck with the fact that appendicitis does not figure as a cause of death. If autopsies were made in the cases in which death is recorded as due to "peritonitis," "inflammation and obstruction of the bowels," etc., I am certain that appendicitis would be found the primary cause of death in a large majority of cases so reported. The honest physician or surgeon who is open to conviction cannot but be convinced of the truth of my statements. One attack of appendicitis is almost sure to be followed by others. Each and every subsequent attack lessens the patient's chances for ultimate recovery —and why? Inflammation of the peritoneum leaves adhesions and inflammatory lymph; leaves an appendix the subject of chronic catarrhal inflammation which forms a fruitful soil for the development of bacterial life. Such an appendix is, in my opinion, the starting-point for a large percentage of the chronic intestinal troubles so commonly seen.

In view of these deductions, and the fact that the mortality of the operation for chronic appendicitis is practically *nil*, I must say that the safest and most logical procedure is operation. The golden opportunity is in the primary attack as soon as the diagnosis is established, thus eliminating the possibility of perforation, gangrene, pus and general peritonitis. Should this opportunity be lost, and the patient recover from the attack, I strongly advise the removal of the appendix as soon as possible.

Name.	Age.	Sex.	Date.	Number of attacks.	Condition of bowels.	Position of appendix.	Pus.	Adhesions.	Manner of treating stump.	Result.	Remarks.
			1890								
D. R.	34	M.	July 20	2	Constipated	S. W.	Yes	Yes	Tied off.	Rec.	Abscess-cavity walled off.
			1891								
A. B.	9	F.	Sept. 12	1	"	N.	"	"	" "	"	Appendix partially gangrenous.
F. F.	28	M.	1892	1	Normal	N.	"	"	" "	"	Abscess-cavity walled off.
			1893								
L. F.	30	M.	April 11	1	.....	N. E.	"	"	" "	"	" " " "
W. I.	35	M.	May 3	1	Constipated	N.	No	Slight	" "	"	Mucous and submucous coats greatly thickened.
R. S. B.	25	M.	May	2	.....	S. E.	"	"	" "	"	Long meso-appendix.
Mrs. K.	25	F.	May	1	....	S.	Yes	"	" "	"	Pain in right iliac fossa since acute attack; in bed for nineteen weeks prior to operation; short appendix.
				7 yr's ago							
C. N.	39	M.	Aug. 14	4	Diarrhea	S.	No	Firm	" "	"	Appendix tightly bound down by adhesions.
E. B. P.	50	M.	Aug. 19	5	"	N. E.	Yes	"	" "	"	Ulceration, with purulent collection in appendix.
Mrs. B.	30	F.	Dec. 29	1	Constipated	S. W.	No	Numerous	" "	"	Appendix gangrenous.
			1894								
T. W.	28	F.	Jan.	2	Diarrhea	S. W.	"	Slight	" "	"	Two coproliths.
A. M.	17	F.	Jan. 2	1	Normal	N.	"	No	" "	"	Appendix post-cecal.
I. R.	21	F.	Jan. 20	1	Constipated	S.	"	Slight	" "	"	Long meso-appendix.
L. V.	49	F.	Jan. 30	3	Diarrhea	S.	"	Firm	Circular amputation, stump invaginated.	"	Appendix short, bound down.
M. C.	23	F.	Feb. 8	1	....	S. E.	Yes	Slight	" "	"	Appendix thickened.
C. F. Z.	23	M.	Feb. 8	2	Constipated	S. E.	"	Firm	Tied off.	"	Adhesions to omentum.
J. McC.	19	M.	March	3	.....	N.	"	"	Circular amputation, stump invaginated.	"	Appendix bent, twisted, gangrenous.
S. F.	30	M.	March	4	.....	N. W.	No	"	" "	"	Adhesions entirely surrounding appendix.
M. S.	60	F.	March	1	.....	S. W.	"	Numerous	" "	"	Long meso-appendix.

M. L.	65	M.	March	1	Constipated	S. E.	Yes	Firm	"	"	"	Short " "
J. T. B.	34	F.	April	1	Diarrhea	S. W.	No	"	"	"	"	Appendix tied down.
R. D.	18	M.	April 3	3	"	N. W.	Yes	"	Tied off.		"	Sinus in right lumbar region leading to orifice in appendix.
H. E.W.	26	M.	April 4	4	"	N. E.	"	"	Circular amputation, stump invaginated.		"	Tip adherent to cecum, ulceration at point of contact; stump with margin of cecal ulcer invaginated.
L. W. G.	31	M.	April 7	1	Constipated	N.	Yes	"	"	"	"	Appendix enveloped in wall of omentum.
E. B.	27	F.	April 17	3	Diarrhea	S. W.	No	No	"	"	"	Appendix very long.
L. P. B.	38	M.	April 18	1	.....	N. W.	Yes	Firm	"	"	"	Appendix twisted and gangrenous.
H. G.	58	M.	April 21	3	.....	S.	"	No	"	"	"	Abscess-cavity walled off.
H. O.	35	F.	April 29	2	Constipated	S. E.	No	Numerous	"	"	"	Appendix enlarged.
L. C.	53	F.	April 30	1	Diarrhea	N.	"	Slight	"	"	"	Walls infiltrated.
E. T.	14	M.	May 3	1	Constipated	S.	"	Firm	"	"	"	Appendix adherent to mesentery; during acute attack passed cast of appendix.
G. G.	50	M.	May 16	10	"	N. E.	"	Yes	"	"	"	
S. C.	22	F.	June 4	2	Diarrhea	E.	"	"	"	"	"	Pain referred to left side, subjective symptoms in right iliac fossa; tip of appendix attached to peritoneum to left of median line.
H. P.	37	F.	May 24	5	"	N. E.	"	"	"	"	"	
Mrs. B.	34	F.	May	1	Constipated	S.	Yes	No	Tied off.		"	Long meso appendix; appendix contained pus.
M. E.	25	F.	May	1	"	S. W.	No	"	Circular amputation, stump invaginated.		"	Appendix thickened at distal end.
R. C. Y.	26	M.	June 7	3	"	S. E.	"	Yes	"	"	"	Tuberculous.
R. S.	19	M.	June 12	1	Diarrhea	S. E.	"	No	"	"	"	Appendix had sloughed.
Mcl.	30	M.	June 10	1	Constipated	N. E.	Yes	Yes	Tied off.		"	
J. M.	26	M.	June 16	4	"	N.	No	No	Circular amputation, stump invaginated.		"	
J. McF.	27	M.	June 25	9	"	S.	"	Many	"	"	"	First attack fourteen years ago; suffered since with entero-colitis; disappeared since operation.
J. H. B.	45	M.	June 27	1	"	N. W.	"	No	"	"	"	Meso-appendix gangrenous.
R. M.	30	M.	June 28	3	"	S. E.	Yes	Yes	Tied off.		"	Omentum gangrenous, tied off and removed.

Name.	Age.	Sex.	Date.	Number of attacks.	Condition of bowels.	Position of appendix.	Pus.	Adhesions.	Manner of treating stump.	Result.	Remarks.
			1894								
E. C.	21	M.	June 26	1	Normal	N.	No	Yes	Tied off.	Rec.	
J. D.	17	F.	July 3	1	Constipated	...	Yes	"	.....	"	
E. C.	26	F.	July 11	2	"	S.	"	No	Tied off.	"	
C. S. B.	22	M.	Aug. 5	7	Normal	N.	No	Yes	Circular amputation, stump invaginated.	"	Appendix much twisted; post-cecal.
M. G.	12	F.	Aug. 9	2	Constipated	N.	Yes	"	" "	"	
H. E.	34	M.	Aug. 10	1	"	S. W.	No	"	" "	"	
H. S.	27	M.	Aug. 26	1	Normal	N.	Yes	"	Tied off.	"	Appendix had sloughed from cecum; stump invaginated.
W. S.	18	M.	Aug. 27	2	Constipated	S. W.	No	No	Circular amputation, stump invaginated.	"	
Mrs. W.	24	F.	Aug. 28	2	"	N.	Yes	Yes	Tied off.	"	Invaginated.
Mrs. F.	32	F.	Sept. 4	2	"	S. E.	No	"	Circular amputation, stump invaginated.	"	Appendix long, clubbed at end.
C. W.	22	F.	Sept. 12	6	"	S.	"	No	" "	"	Thickened appendix.
B. McK.	22	F.	Sept. 26	4	"	S.	"	Yes	" "	Died	Adhesions very dense; appendix surrounded by inflammatory lymph; patient died of peritonitis.
J. N.	24	F.	Oct. 3	4	"	S.	"	"	" "	Rec.	Fecal concretion at tip.
J. A.	30	M.	Oct. 6	16	"	S.	"	"	" "	"	Appendix short.
F. N.	22	M.	Oct. 11	numerous	"	S.	"	No	" "	"	Repeated attacks for two years; appendix very long.
N. M.	28	M.	Oct. 17	7	"	S.	"	Yes	" "	"	Appendix completely twisted on itself, free bleeding during operation; glass and gauze drainage.
J. R.	43	M.	Oct. 18	3	"	S. E.	Yes	No	" "	"	
L. F.	23	M.	Oct. 23	3	"	S. W.	No	"	" "	"	
.....	30	M.	Oct. 24	2	"	S. E.	Yes	Yes	Tied off.	"	

By asepsis and careful technique the operation can be done with but little risk to life, as has been proved by such men as McBurney, Richardson, Bull, Fowler, and others. In further support of this I herewith tabulate sixty-one cases of operation for chronic appendicitis, with one death.

DISCUSSION.

Dr. Charles McBurney, of New York: Mr. President and Gentlemen: It gives me great pleasure to be here, and I thank you and Dr. Deaver for the privilege of being allowed to take part in the discussion of the subject of his valuable paper—a subject in regard to which so much of the valuable work that has been done has been in Philadelphia. If my remarks are a little disjointed I hope that you will excuse me, for before Dr. Deaver finished his paper I not only felt quite an uncomfortable sensation in my right side, but I also noticed that a number of the gentlemen about me were palpating their right iliac fossæ. This has a little disturbed the continuity of my thoughts.

I agree so thoroughly with Dr. Deaver in his conclusions that it is perhaps a little unfortunate, as far as the interest of the discussion is concerned, because I cannot conscientiously attack him upon any of the conclusions that he has advanced. If I am to talk to you upon the subject I should like to follow somewhat the same line that Dr. Deaver has taken. I think that it was very well said by Richardson, of Boston, in the last paper he wrote on this subject, that acute appendicitis is the most important acute abdominal disease that is now before the scientific world. This observation to those who have seen a great deal, and are now seeing a great deal, of appendicitis seems a little trite. At the same time it is a statement that is well worth spreading throughout the professional world, especially among those who are scattered in the small places, where the number of cases of any one disease is small, and where a practitioner may have a considerable practice and yet in years not see, or not recognize, a single case of appendicitis. The practitioners of medicine are the ones who need to have impressed upon them forcibly the fact that this is the most important of the acute abdominal diseases. In the larger cities the matter is constantly being brought before the profession, and there are comparatively few medical men who are not more or less familiar with it. I look upon acute appendicitis as "the most important acute abdominal disease which is now before the scientific world," for various reasons. In the first place, on account of its great frequency; in the second place, on account of its great fatality; in the third place, on account of the situation of the lesion; in the fourth place, on account of the extraordi-

nary multiplicity of its pathological processes; and, lastly, on account of the very great range and variety of the symptoms that are presented by the different cases. Moreover, it is an extremely important disease (and I think that the medical men have not given this point full weight), on account of its now well-recognized pathology; and I doubt if there is a gentleman in this room who would seriously claim that any one has demonstrated a plan or method for the *prevention* of appendicitis which in the least appeals to one's scientific sense or which has the least scientific foundation. All these reasons combined warrant the statement as to the importance of the disease.

The mortality of the disease, according to my convictions, based upon the examination of a good many statistics and upon a fairly extensive study of the subject, is, when *all* the cases are treated *medically*, at least 25 per cent.

What is the cause of this large mortality? Is it not the traumatism of the ruptured appendix? Is it not the fact that some interference takes place with the discharge of the secretions in the sense that the body needs these secretions? Is it not the fact that there is displacement or malposition of any of the viscera from inflammatory conditions? None of these things have anything to do with the mortality except as leading to another condition. The real cause thoroughly appreciated by those familiar with the disease is often overlooked, and we hear a great deal about "mild inflammation" of the appendix, and about "catarrhal inflammation," as though it were no more important than catarrhal conditions of the nasal and other mucous membranes. The real source of the mortality is sepsis—the disease is essentially a septic one, and from the very beginning of an attack of appendicitis the individual is attacked with sepsis. In a certain sense he is in a condition similar to that of a person who has received a wound on the end of his finger with a septic instrument. If the individual is especially susceptible to sepsis, —and there is an enormous difference in individuals in this respect—he is extremely liable to go on to the full development of general sepsis. If he is little liable to sepsis, and the local conditions permit of rapid subsidence of the inflammation, the attack is spoken of as a mild attack of catarrhal inflammation. If the wound is on the finger the story is similar. Some individuals become enormously septic from a small wound, while in others the wound will rapidly clear itself, and the individual will escape from everything but the local signs of sepsis; but look at the difference between the two lesions. The one on the finger can be readily cleansed and the best surgical treatment can at once be applied, and who would think of treating a septic wound of the finger medically? I would ask the medical profession what remedy they possess in the whole pharmacopœia which has the least effect in controlling the course of a case of appendicitis? I know of none. I know of no demonstration that shows that any medical remedy has had any effect on a case of appendicitis. I am perfectly willing to admit that plenty of cases of appendicitis subside after being quite violent, and even alarming, under certain conditions, such as the application of cold or the administration of a cathartic;

and the choice among cathartics is varied. One says castor oil, another prefers salines, and another calomel. And I have had different gentlemen in turn tell me that each one of these remedies was *the* remedy. I have never seen a case, or the history of a case, by which I was in the least convinced that medication had had any influence on the disease. This is a very important consideration. If I am wrong, I should like to be corrected. I have not seen any argument that would lead me to take any other view than the one I have expressed. The argument that persuades me that I am right, or nearly right, is this: I have had numerous cases that have come into the hospital, and which I have kept under careful observation, and on which I have not operated, for I do not operate on all cases. I prefer, where I think it is safe, to allow an attack of appendicitis to subside, and operate after the attack is over, in the period of quiescence. Having these cases under observation, and allowing the acute symptoms to subside, and doing absolutely nothing, except to enjoin rest in bed, I find that they do as well as the patients who have been given castor oil, salines, or calomel. I am satisfied that special medical remedies have absolutely no effect in controlling the disease.

I think that one might readily admit the truth of the statement that I have just made, if he were ready also to admit the etiology of the disease, which has already been referred to by Dr. Deaver. If, without taking too much time, I were to state what I believe to be the real cause, the originating cause of disease of the appendix, I should say that it was interference with its proper drainage. That is putting it too briefly. I look upon interference with the proper drainage of the appendix as regards its effects on the mucous membrane precisely as I look upon stricture of the urethra as affecting the mucous membrane of the urethra behind the stricture. We know that in the appendix, as well as in the intestines, large and small, we have a prodigious quantity of bacteria, particularly the bacterium coli commune. We know that health continues with these bacteria there. If the mucous membrane is healthy, no disturbance of the normal condition takes place. We know that a man may have a cystitis of a very virulent type, and have it for years, and if there has been no disease of the urethral mucous membrane primarily, he will pass septic urine without inducing any disturbance of health of the urethral mucous membrane. If that man has a moderate stricture, with the calibre of the urethra reduced from one to three sizes only of the French scale, immediately changes in the mucous membrane behind it commence. The fluids that formerly passed over it without harm become very septic, and the individual is liable to extension of the septic disease, and it is not uncommon to have a stricture in a few days diminish very rapidly in size, with ulcerative processes, and even gangrene, occurring. I fully believe that a somewhat similar process occurs in connection with the appendix. If there is any obstruction to the escape of the normally harmless contents of the appendix, and this interference may be

caused by a colon distended with gas or feces, by displacement of the viscera, by accumulation of fat in the mesentery, turning it to one side, by the entrance of a little soft feces into the mouth of the appendix, or by a hard concretion or, rarely, a true foreign body—anything, either from within or without, which interferes with the calibre and prevents normal drainage at once establishes conditions which are apparently all that are needed to encourage the bacteria of the appendix, to multiply and to take on a septic power which they did not primarily have. That, it seems to me, is the explanation of all cases, and I think that numerous specimens that I have handled confirm this view. Where the appendix was not completely destroyed I have never failed to find at some point or other a definite interference with the calibre of the tube, and beyond this point the disease. In every case of disease of the appendix the important element is the septic process, and the extent of this is not clearly indicated by the symptoms in the early stages of the disease, so that it is extremely difficult for the most experienced to lay out a prognosis, and this is where we meet with the real trouble in deciding the question as to the method of treatment or the time of operation.

A great deal is said in opposition to surgical treatment, based on the fact that cases do spontaneously recover. This is constantly being referred to. You will see statements made by physicians, and even surgeons, that, according to their experience, all of these cases get well. How do these cases get well? I have seen cases recover without treatment, but in using that phrase I should like to define what I mean by "getting well." An individual may have a mild condition of septic infection, and the worst that it does is to cause swelling of the mucous membrane. This may subside and the sepsis disappear, and the individual lose all signs of disease and be apparently well. Some of these cases will remain perfectly well, because the effect on the mucous membrane has not been sufficiently grave to induce any important change in structure. A large number of cases which are spoken of as successful recoveries are cases which have gotten over a particular attack. They do not have any symptoms, and they feel well for months, perhaps. Then they have another attack. This occurs in almost all. It may not be for several years, even ten years. Some of these cases which for a long time have been looked upon as well have a second and fatal attack. I have known a number of such cases—quite large enough to establish the belief that cases that have had one attack are not in any way to be trusted. Again, in other cases that "get well" a tumor of considerable size forms, and the patient goes through a severe illness, a great deal of anxiety is felt as to the result, and the physician becomes quite alarmed. Suddenly the patient begins to feel better in the course of an hour or over night. It is then noted that the abscess has opened into the intestines, and often everyone interested congratulates himself that the patient has escaped the knife and that he has gotten well. That patient is not well. The condition left is that of an empty sac lined with granulation tissue emptying into the intestine. Almost the whole appendix may be left. After a time this

patient has another attack of pain, and another abscess forms and discharges in the same way, or it may rupture into the peritoneal cavity. I have seen this repeated over and over, so that I claim that this method of getting well is a very poor one.

Now, if we have no medical treatment that controls the disease—and I maintain that we have none—we are not going to allow these patients to present symptoms of serious illness without applying any remedy. Have we any remedy? Unquestionably; a beautiful remedy, one of the most perfect, clean-cut, and complete remedies which was ever applied to the treatment of disease. There is no remedy that compares with it, provided it is applied at the right time. The *right* time is before the pathological processes have done much harm. An appendix that is inflamed and contains a little pus has in the majority of cases not suffered much harm. There are, however, individuals who will be generally septic before the appendix shows anything but the beginning signs of disease. These individuals are those who become septic on very slight provocation. With these exceptions the appendix will stand a great deal, and up to twelve, twenty-four, or forty-eight hours the disease will be found confined to that organ. That is the time to operate. The reasons that I should especially urge in favor of this are these: The operation is very free from danger. This may seem a rather strong statement, but I assure you that it is true. I have never yet seen a patient die after an operation for acute appendicitis done at what I call the proper time. I do not mean by that to exclude all the cases that die. Where the appendix is not gangrenous, or where the pus has not extensively involved the pelvis, or traveled over to the other side or very far upward, the cases get well after operation. There is no difficulty in the operation at this stage in a first attack. The appendix is readily separated, there are no firm adhesions, the area of disease is small, the wound made is small and readily closed, and the healing of such wounds is extremely perfect. These are the reasons why I urge early operations. There is practically no mortality in the early operation except in the cases spoken of, where the patient will be septic from the very beginning.

What is the advantage of the early operation over the later operation? Where the patient escapes the early dangers of sepsis the disease may go on to the formation of an abscess, and this used to be looked on as a satisfactory termination. It used to be thought that if you could have a nice abscess presenting well toward the outer part of Poupart's ligament you were in a good position, because any one could open that and let out the pus. When a large abscess is formed, to be properly treated, it must be laid freely open. There is a large wound which must be treated widely open in order to obtain complete healing. Under the most careful treatment not a few of these cases continue to have a sinus running down among the intestines. Some continue for years, some never get well, in others there are extensions from the sinus,

and the patient finally dies. All this is avoided by the early operation. Moreover, the cases of late operation for large abscess are those which are apt to have subsequently large ventral hernia.

If I have not outspoken my time I should like to say a word as to the reason why operation is often deferred until a late period. Why is it that we are so often called to operate on an abscess ten days, two weeks, or a month old? Why is it that we are so often called for the first time to see a patient who is said to have appendicitis, and when we see him find the patient in an advanced stage of septic peritonitis? What is the reason for this delay? The reason is unquestionably the perfectly unfounded belief in the value of certain remedies, including time and opium. The physician when called often does not recognize the character of the disease, and administers some preparation of opium, causing a delay of at least twenty-four hours. When the effect of this has worn off, the patient insists on another dose. This keeps the patient comfortable, and often causes a fatal delay in the application of the real remedy.

Another reason of delay, and one which the surgeon must combat, is the widespread belief that the disease is not such a very dangerous one, and that the physician can afford to wait three or four days, and that *he* will decide when the time for operation has come. There is no reason why the physician should decide in a case of appendicitis, or in any other surgical case, when the time for operation has come. The proper one to decide this is the man who has studied not only the ordinary external appearances, which the physician also studies, but who has also seen the lesions and compared the signs with the lesions. He is the one to decide when the time for operation has come in any given case.

Dr. H. A. Hare: I should like to ask a question in regard to the administration of a purgative in these cases. I should like to hear Dr. McBurney's opinion as to the advisability of administering them prior to operation. The old idea that the head of the colon contains hardened feces has been exploded. If this is the case, I cannot see that much is gained by the administration of a purgative which, theoretically, may sweep feces into the peritoneal cavity through an opening in the bowel. If the cases are surgical almost from the start I think that unless the intestinal canal were loaded with feces it would be better to operate at once than to lose time by the administration of a purgative and take the risk of having fecal matter swept into the peritoneal cavity. I would ask if any of the surgeons have met with cases of perforation where the use of a purgative has apparently caused the escape of fecal matter into the peritoneal cavity?

There is another point that I did not understand clearly. From the general tenor of Dr. McBurney's remarks I infer that he believes that these cases should be operated on very early and without delay, but I also understand him to say that, as a rule, he prefers in a first attack to wait until the symptoms have moderated and the acute inflammation has passed away. If the

appendix is as septic as he believes, it seems to me that we should operate at once without delay. A delay of twenty-four hours may be fatal.

Dr. John Ashhurst, Jr.: I suppose that I occupy the position of one of those who have been somewhat pityingly referred to by Dr. McBurney as conservative surgeons somewhat advanced in years.

A good deal has been said as to the difficulty of diagnosis in cases of appendicitis. I do not profess to be more skilful than others, but I think that the diagnosis can usually be made by surgeons used to the employment of the sense of touch, and I believe that it may equally be made by physicians who have educated the same sense. I am not prepared to go as far as has been done by some writers in claiming that appendicitis is a disease which should necessarily be treated by a surgeon from the beginning, for I believe that in a considerable proportion of cases—indeed in a large majority, if judiciously treated from the beginning—the patient will get well without surgical interference. At the same time I would say that the physician who has not that delicacy of touch which will enable him to recognize the local condition, or to detect those changes which precede the formation of pus, should have associated with him a surgeon from an early period in the case.

Dr. McBurney has asked if there is any medicinal treatment which can effect a cure in these cases. I would remind him that when we speak of curing a patient we mean simply that we take care of him and that the patient gets well. I do not expect to convince Dr. McBurney that it is in the power of medicinal remedies to cure appendicitis; but the fact is undoubted that under the use of medicinal treatment, with the simple local applications which physicians are in the habit of making, many cases of appendicitis will get well and remain well for an indefinite period. Some remain well for the rest of their lives. Others have a second attack after a longer or shorter period. It is the custom with advanced surgeons at the present day to fix a limit for disease, and to say, for instance, when a cancer of the breast which has been removed comes back after three years, that it is not a recurrence, but a new cancer; I am surprised that the same surgeons will not acknowledge that appendicitis may be cured, and are unwilling that, if it return after a period of even ten years, it should be considered as a new case. The treatment which I have adopted in the early stage of appendicitis has not been the administration of purgatives or of occasional doses of morphine, and I have no doubt that Dr. McBurney is right when he says that it is better that the patient should be left in bed without any treatment whatever than that he should be given repeated doses of purgatives or only occasional doses of morphine to relieve pain. The plan which I have found successful is that recommended many years ago by the late Dr. Alonzo Clark in the treatment of peritonitis—that is the administration of opium systematically until the respiration is brought down to twelve in the minute. I combine the opium with belladonna, and under this plan I have seen in a considerable number of cases cures of appendicitis as of other forms of peritonitis. As to "masking

the symptoms," although the opium relieves the pain, yet I think that the skilful physician or surgeon can ascertain whether or not the patient is doing well from other signs, such as the temperature range, the local symptoms, the amount of movement of the abdominal walls, etc. In cases occurring in robust persons I add the use of leeches to this treatment. I believe that life has been saved by the application of leeches in the early stage of appendicitis as well as of other forms of peritonitis. When this mode of treatment does not succeed I think that operation is indicated.

As to operation after the patient is well, undoubtedly it is a much simpler procedure, and the prognosis is better than in acute cases. At the same time, I have never felt justified in resorting to the operation in the interval, except under special circumstances. I have operated in five such cases, and all the patients have recovered. I have operated also in a number of cases of acute appendicitis, and, while I have lost some patients, the majority have gotten well. I may say with Dr. McBurney that the fatal cases were septic from the beginning, and were cases in which death would have occurred under any circumstances. I have never regretted operating for appendicitis, and I can say most decidedly that I have never regretted declining to operate. I have seen cases, such as Dr. Deaver mentions, where pus was free in the peritoneal cavity, and in some of these I have succeeded in saving life by operating as a last resort.

I have seen some curious things in operating for appendicitis. On one occasion I saw what I at first thought was an unusually long appendix, but on drawing it out it proved to be a round worm, free in the abdominal cavity. The patient recovered.

My belief, then, is that a large majority of patients with appendicitis will get well without an operation, if judiciously treated. If an operation is required, I believe that still the majority will get well. The patients who do not get well are those in whom there is what may be called a septic diathesis, where the patient will become septic from slight causes. The majority of these will die, although a few may by operation be snatched, as has been said, "from the very jaws of death."

As regards the propriety of an operation for fear that the patient may have another attack, perhaps many years afterward, that is a suggestion of what I have called advanced surgery which I am hardly prepared to accept. It is somewhat analogous to the recommendation of removal of both testes in elderly men, from fear that at some subsequent period they might have enlargement of the prostate.

Dr. A. V. Meigs: Dr. Deaver has stated that the diagnosis is easy to make in appendicitis, and our guest from New York alluded to the many pathological lesions which occur as consequences of appendicitis; but he said nothing of the diseases of the peritoneal cavity in which appendicitis may occur as a secondary consequence. During the past summer I have had two abdominal operations done at the Pennsylvania Hospital. The first case was

that of a woman I watched a day or two, and then had our gynecologist see with me, to try to ascertain the nature of the disease. We were unable to decide whether the attack was one of appendicitis, or if the disease had begun in the tube or ovary. As the woman's condition became almost desperate, it was decided to operate.

The appendix was normal, and the only lesion found was a dermoid tumor of the *left* ovary—the side opposite to the apparent seat of disease previous to operation. This tumor was removed, and twenty-five hours later the woman died. At the post-mortem examination nearly two hours were consumed in ascertaining that the real cause of the trouble was an abscess underneath the liver, which had ulcerated through the gall-bladder, so that bile and pus were escaping into the peritoneal cavity. This condition was not even suspected at the time of operation, nor could it have been found, or, if found, relieved.

During the twelve or thirteen years that I have been physician to the Pennsylvania Hospital I have seen a great many post-mortem examinations, and I can remember only one case of perforated appendix. I do not believe the lesion is so common as some would lead us to believe.

Last summer I had a patient in the hospital with all the commonly-accepted signs of appendicitis—obstruction of the bowel, tympanites, pain, and tumor in the right iliac fossa. He went from the hospital apparently quite well. The treatment was a quarter of a grain of opium and one-twelfth of a grain of extract of belladonna every two hours for a few days, and then small and repeated doses of vegetable cathartics. Castor-oil, salines, and calomel merely induce watery movements and do not empty the bowel of feces.

Dr. McBurney has said that persons with appendicitis do not get well, but only improve temporarily, and then suffer with relapses. One's personal experience cannot, in the nature of things, be very large, but I can recall several persons who have had the disease and have been fifteen and even thirty years without any recurrence.

The last case to which I shall allude is the second one I had operated upon in the hospital this year. I should like, however, to take exception to the statement that physicians are not willing to have surgical opinions in obscure cases of abdominal disease; on the contrary, I believe they eagerly seek such help. My patient was a boy of eleven, who had been in the hospital four months previously, under Dr. Ashhurst's care, with an attack of general peritonitis, which came on from his having been kicked in the abdomen by another boy. He recovered, but returned to the hospital in September with symptoms of intestinal obstruction. A number of physicians and surgeons saw him, and there was doubt in regard to the diagnosis, and even difference of opinion as to the treatment. Some wanted salts administered, but this I was unwilling to agree to, and would not do so until at last I was overruled, as it was thought necessary that he should take salts before any operation was done. At the consultation to decide the question of

operation, I opposed it upon the ground that the diagnostic indications were too obscure, and that it was too late. In less than twenty-four hours the patient took four and a half ounces of epsom salts, almost entirely without purgative effect. At the operation, when the appendix was first seen, everyone agreed that it was not diseased and it was dropped back into the abdomen. Later, in the course of the manipulations of the surgeon, some pus welled out of the abdominal incision. The appendix was again drawn to the surface, said to be diseased, and removed. After forty-eight hours the patient died. At the post-mortem examination it was found that there were many adhesions of and around the caput coli and a small pus deposit behind it. Where any of the adhesions were torn loose it was found that at the point of adherence there was some fresh yellow lymph or pus. The appendix, when opened, appeared quite natural, and the only sign of disease about it was one of these small spots of yellow lymph or pus upon one side of its exterior surface, where it had been adherent. The real cause of the obstruction of the bowel was found to be an old adhesion of ileum. A portion of the ileum, about eighteen inches from the ileo-cæcal valve, was dragged from the left side of the abdominal cavity, where it naturally lay, and fastened by an old thick adhesive band in the right iliac fossa. So firm was the adhesion and so far had the knuckle of intestine been dragged from its natural position that the obstruction was practically complete so far as concerned the passage of solid material. This, a consequence of the attack of peritonitis four months previously, was the true and only cause of the final attack, and the adhesions and pus about the head of the colon and appendix were all secondary to it. It is absurd to suppose that a surgeon at an operation would, with two fingers in the abdominal cavity, have differentiated all this, when it took nearly two hours to come to an understanding of it at the post-mortem examination. The diagnosis in many cases of obstruction of the bowel is most difficult if not impossible. One point more: In this case the bowel above the point of obstruction contained almost no feces, while below the obstruction there was a great deal. If the salts given had operated, the surgeons would have thought the obstruction had yielded. This seems to be another argument against the much-vaunted modern treatment of obstruction of the bowel with salines.

Dr. William Osler: At the Johns Hopkins Hospital my orders are not to admit cases of appendicitis to my ward, but to give the surgeons the responsibility at the outset. Humility always has its reward, and in consequence I am now often called upon by surgeons to say whether or not operation shall be performed, and more than once in the last three years have I told a surgeon to stay his hand, as in all probability the attack was not appendicitis. One case proved to be hysteria, and another ovarian trouble. The chief difficulty lies in the early recognition of appendicitis. I should like to ask Dr. Deaver in particular, and surgeons in general, how many appendices are removed that are about as normal as the tubes and ovaries which we used

to see from the gynecologist. Believe me, there are appendices removed that are not in a septic condition. I have seen them. I admit that a man is better without the appendix than with it, and I do not quarrel with any surgeon for removing the appendix. I quarrel with them for scolding the physician for any delay. Physicians nowadays are wider awake in regard to this disease and more willing to hand the cases over to the surgeons at an early date than formerly.

I cannot agree with the statement that Nature never completely heals disease of the appendix. Some years ago I reported my cases of appendix lesion in eight hundred autopsies. There are several in which unquestionably the appendix was completely and entirely healed, being converted into a fibrous cord.

Dr. George E. Shoemaker: I would ask Dr. Deaver in closing the discussion to bring out one point more fully. He said that he made a routine practice of giving a purgative, and if the patient did not improve, he operated. I should like to know how many cases he has met in the period covered by his paper where he gave a purgative and operation was not needed. I should also like to know how many cases he found so ill that he declined operation.

Dr. John B. Roberts: Some observation and some experience have led me to the conclusion, I think, an irresistible conclusion, that at the present time, the appendix is being removed more frequently than pathology demands or good surgery justifies.

Dr. S. Weir Mitchell: It is, perhaps, scarcely fair to criticise after results of operations of necessity, nevertheless, it is clinically interesting to know that the removal of the appendix is occasionally followed by conditions of general discomfort, and occasionally by attacks of local pain, which show that the operation is not always without unpleasant consequences. Less stress has been put upon the use of ice than I expected. I doubt very much whether the chilling influence of ice ever extends as far inward as the part diseased; but here, as elsewhere, the use of ice is followed by reflex effects upon the bloodvessels far beneath the surface.

Dr. J. C. Wilson: It has been my good fortune to see many of the cases in the hands of Dr. Deaver, my surgical colleague at the German Hospital. As a medical man, it occurs to me that perhaps the diagnosis of appendicitis is not always so simple, the course of the affection so definite, nor its treatment so clear as would appear from the statements of the surgeons.

It has been my experience to occasionally meet with a disease presenting the early symptoms of appendicitis, but not followed by the near or remote grave consequences upon which so much stress has been laid in the discussion to-night, and after the lapse of an indefinite period, in some cases now measured by many years, not recurring.

The attacks occur oftenest in young adults, but also later in life. They follow an indigestion or exposure to cold. There are colicky pains, tender-

ness in the region corresponding to the appendix, a disposition to flex the thigh, mild fever in some instances, sometimes none at all. Under various forms of treatment, sometimes calomel, sometimes salines, sometimes even opium, recovery takes place in from thirty-six to forty eight or seventy-two hours, a recovery which, as I have said, appears to be permanent. Does this condition fall properly under the head of appendicitis or is it a non-infective process of an essentially different character from that which has been described so admirably to-night? If so, what are the criteria by which a differential diagnosis can be made and how are we to at once recognize the cases in which the early operation is necessary to avert the most serious consequences, even to save life?

Dr. Deaver: As to the diagnosis, with few exceptions I have had no trouble in making it. Where we have a history of acute indigestion with abdominal pain, paroxysmal in character, soon localizing itself in the right iliac fossa, with tenderness at McBurney's point, in nine cases out of ten the case is one of appendicitis. It seems so simple that I am surprised to hear medical men speak of the difficulty.

The question has been asked how many normal appendices have been removed. I have not removed a single one. The great bulk of these cases had extensive adhesions, many contained pus in the appendix or its neighborhood, others had pus between the layers of the meso-appendix. A large number contained the bacillus coli communis, and a number the streptococcus.

Dr. Osler speaks of having seen cases where the appendix had entirely healed. These cases have been well described by Dr. Senn as obliterated appendices. I have seen this condition present with pus around the appendix.

The object in using a purgative is to clear out the intestinal canal, which probably contains irritating matter as a result of the indigestion. It should be given early. I have seen a purgative administered late followed by the escape of fecal matter through a perforated appendix. I have seen this accident follow the use of an enema in a case where opium had been given. I operated in that case against my will, and found the belly cavity filled with the injection. In all my cases the purgative is given to clear out the alimentary canal and not for a curative effect. In acute appendicitis the purgative is given for the removal of foreign matter; then, as the case progresses, it is evident that the trouble is not due to the presence of indigestible material.

My experience agrees with that of Dr. McBurney that where I have operated in acute appendicitis, at what I considered a favorable time, I have not lost a case. That is a strong argument in favor of early operation.

The question of the recognition of pus. It is unfortunate to allow a case to go that long. I was called to-day to see a case where the patient was expectorating fecal pus. That is one of the cases where the appendix occupied a position behind the colon, between the layers of the meso-colon, and abscess formed and was not evacuated,

The case to which Dr. Meigs referred, where there was pus in the neighborhood of the appendix, I believe, in all probability, was a case of appendicitis, and that the diffused peritonitis and formation of adhesion were the cause of the kink in the bowel. I have seen, in a number of instances, secondary abscess of the liver as a result of appendicitis. I have also seen abscess of the mesentery.

I do not operate on all cases of acute appendicitis I see. Many of these cases of simple appendicitis are amenable to treatment.

Dr. McBurney: I think that Dr. Ashhurst rather misunderstood my position in regard to the class of cases on which I would advocate operation. I do not believe in operating on every case at once, nor do I believe that there is any special danger in a certain amount of delay in a considerable proportion of cases. I think that many cases will allow of deliberate study before any decision is reached. I do not operate immediately on any patient whom I do not consider *likely* to become very ill. Surgery has reached such a point that it is better to operate before extreme illness than after this condition has appeared. If the patient is not very ill, and can be seen every few hours, I am willing to postpone the consideration of operation. Many of these cases improve, and then the question of future course arises. I tell them that the probability is that another attack will occur, and advise them to reside near good surgical assistance in case further trouble should occur. If a second attack occurs in a short time, the question is probably settled, and the appendix should be taken out. Where the patient is about to change his residence to a place where he could not be promptly seen by a surgeon, I have advised the operation in many cases, and have never regretted it. In some cases, where such advice has been neglected or not sought for, another attack has occurred with a fatal result.

With regard to cathartics, I have mentioned that I never use them. The reason is that in not a single case on which I have operated have I found feces in the caput coli. If there are no feces in the neighborhood of the appendix I do not care to use cathartics. I seldom give opium, because I do not wish to mask symptoms, and I do not believe that it does any good. One great objection to the early administration of opium is that it leads to delay in making a careful examination and diagnosis. After the diagnosis is made I see no objection to giving a moderate dose of morphine to obtain partial relief when pain is really severe.

THE WORK OF THE GYNECOLOGICAL CLINIC OF THE HOSPITAL OF THE UNIVERSITY OF PENNSYLVANIA, 1893–1894.

By CHARLES B. PENROSE, M.D.,
PROFESSOR OF GYNECOLOGY IN THE UNIVERSITY OF PENNSYLVANIA.

[Read November 7, 1894.]

I HAVE presented this paper to the college not to give a number of statistical tables and reports of cases, but because several general conclusions can be drawn from the work of the clinic, which I hope will be of interest to others than mere specialists in gynecology.

The class of cases which seems best suited for the instruction of a large number of students are those which require the operation of cœliotomy. And for this reason nearly all the operations performed at the gynecological clinic have been of this character. The various plastic operations upon the perineum, the vagina, and upon the uterus through the vagina, can be seen with advantage by but a few students at one time, and, consequently, all these minor operations are reserved for ward-class instruction.

Many physicians and a good many students, at first, have the impression that a clinic at which repeated cœliotomies are performed presents nothing of interest beyond the mere details of the operation, and that instruction in such clinics covers only the general and special operative technique.

This is a mistake. The instruction in the operative technique of cœliotomy is of very minor importance to undergraduate students; and cannot compare in value to the instruc-

tion derived by viewing, through a cœliotomy, diseased structures in place, or their examination when fresh, immediately after removal. The students have an opportunity of observing the history of the patients before the operation—in many cases of examining them in the ward-classes beforehand—and after this introduction the study of the pathological specimens removed at the clinic has much increased interest and value; confirming or upsetting the diagnosis, and shedding light on all the objective and subjective symptoms which the patient had presented.

A clinic of this kind instructs in a way analogous to an autopsy. The special technique of the operation is of value only to specialists. It is remarkable what a great variety of diseases of women are presented for study through the operation of cœliotomy even in a limited number of cases. Forty-six cœliotomies were performed at the clinic last winter, and these are some of the diseases which were studied: Extra-uterine pregnancy; oöphoritic, paroöphoritic, and papillomatous cysts; all the various forms of ovaritis and salpingitis; tuberculosis of the internal genitals; fibroid disease of the uterus; myxomatous degeneration of the uterus; cancer of the uterus, both of the fundus and cervix, etc.

Clinical instruction by means of cœliotomy is not given at the expense of an increased mortality. The mortality, after cœliotomy, in the general clinic-room of a clean hospital is not necessarily any greater than the mortality in the best-equipped private operating-room. I am aware that there is still a prejudice in the minds of many physicians against the performance of the operation of cœliotomy in the general hospital clinic, but I feel convinced that this prejudice is unwarranted at the present day.

A few simple preparations will render the clinic-room as suitable for an aseptic operation as any place can be made; and the presence of a number of unsterilized medical students does no harm as long as they and their dust are kept from contact with the patient.

Infection undoubtedly takes place from contact. Infection

from the atmosphere must be very rare. My series of forty-six cases, though small, still goes to sustain this view. For in this number there was no case of sepsis or peritonitis. The only death which occurred followed almost immediately after the operation, from shock and hemorrhage, in an incompleted operation for a large, densely-adherent, retro-peritoneal dermoid.

The aseptic technique is very simple and easily followed. Before the clinic, the amphitheatre is scrubbed, and the railing separating the amphitheatre from the benches is covered completely with large cotton sheets wrung out of a bichloride solution. All the tables, stands, and chairs are covered in a similar way. The operator and assistants are clothed in white cotton shirts and trousers, which are washed for every clinic. Operator, assistants, and nurses are covered with large sterilized aprons. Dressings and silk ligatures are sterilized in a steam sterilizer, and the instruments are boiled in a solution of bicarbonate of sodium. Distilled water is used for all purposes throughout the operation.

No one is permitted in the amphitheatre except those persons who are directly engaged in assisting at the operation, and these are limited to the minimum number. The wound is usually closed with buried sutures—of silk and catgut—and with an intra-cutaneous suture of the skin. By such simple means perfect asepsis can be attained.

I have found that the practical and scientific work of the clinic is very much assisted by having facilities for making bacteriological examinations on the spot during the operation. And it seems that such association of bacteriology and surgery would be of much advantage in other branches besides abdominal surgery.

In the gynecological clinic the question of drainage after cœliotomy is—except in rare cases, as of suspected hemorrhage—determined absolutely by bacteriological examination of any suspicious fluid which may have escaped into the peritoneal cavity. The result so far, not only at the University of Pennsylvania, but outside, has confirmed my faith in this method.

These bacteriological examinations have shown that the pus

in very many cases of pelvic abscess—of ovarian, tubal, or some other origin—contains no micro-organisms; it is sterile and harmless, incapable of producing sepsis or peritonitis. The pus containing gonococci alone, at least in limited numbers, seems also to be harmless when brought in contact with the peritoneum. The practical value of this application of bacteriology is very great. It enables us to follow a median course between the extreme advocates of drainage in cœliotomy and the extreme opponents of drainage; and the course is determined not by prejudice, but by scientific reasoning. In all cases where the bacteriologist finds no micro-organisms in the purulent or other material which escapes into the peritoneum, the abdomen is closed without irrigation and without drainage. When staphylococci, streptococci, or the bacterium coli commune are found, both irrigation and drainage are employed.

The examinations are made very quickly—within five minutes—and in no way interfere with or delay the progress of the operation. It is very inaccurate to speak of flooding the pelvis and abdomen with pus and of saving the life of the woman by irrigation and drainage, simply because a sac containing purulent-looking material has been ruptured during removal. The purulent material is only dangerous if it contains septic or pathogenic germs, and the surgeon has no way of determining this fact without the aid of bacteriological examination. Most of the irrigation and drainage which is done in a routine way without such examination is unnecessary, and consequently harmful.

The result of the clinical experience of the past winter is to confirm my opinion of the value of the procedure of removing the uterus in many cases in which formerly we had been contented merely with the removal of tubes and ovaries. In cases of pelvic abscess it enables us to make the most complete removal of the abscess sac and to perform a neat, clean operation; and in many cases of ectopic gestation and pelvic inflammatory trouble it enables us to check hemorrhage—otherwise very embarrassing.

In septic cases accompanied by disease of the endometrium the relief is immediate and permanent.

The operation of ventrofixation or suspension of the uterus has also given most satisfactory results in retroversion, retroflexion, and prolapse of the uterus.

A subject of special investigation last winter was that of tuberculosis of the Fallopian tubes. All the Fallopian tubes and ovaries removed for the general condition designated pelvic inflammatory trouble were submitted to a most thorough microscopical examination by Dr. Beyea, who found a large proportion of them tuberculous. The number of cases is too small to warrant the assumption that this proportion will be constant in any series of similar cases. It is, however, greater than any yet reported. We found that in our series of pelvic inflammatory cases 20 per cent. were tuberculous.

The tubercular process seems to begin in the distal third of the Fallopian tube, and thence to spread to the ovary and peritoneum or to extend to the uterus. The results in the tuberculous cases were all good and seem to be permanent. The relief from pain was immediate, and there has been progressive increase of weight and strength.

It is impossible to tabulate, in a perfectly satisfactory way, any series of cases of pelvic disease in women, because we always find a variety of pathological conditions together. In the preceding series I have divided the cases according to the form of the operation, and have mentioned the most conspicuous pathological condition present.

The cases of hysterectomy for salpingitis and endometritis include those cases in which there is purulent inflammation of the Fallopian tubes, generally of gonorrhœal or post-puerperal origin, with chronic purulent endometritis and a friable degenerated condition of the body of the uterus. The operation of hysterectomy is a complete removal of all diseased structures. After simple salpingectomy in these cases the woman continues to suffer for an indefinite period with a virulent form of leucorrhœa, which, if of gonorrhœal origin, may not yield to any plan of treatment.

Cœliotomies Performed at the Gynecological Clinic of the Hospital of the University of Pennsylvania. (1893–1894.)

Hysterectomy—Intra-Abdominal Method.

	Number of Cases.	
Fibro-myoma	3	
Calcareous degeneration of fibroma	1	
Salpingitis and endometritis	6	
Extra-uterine pregnancy	1	
Cancer of cervix and body of uterus	1	
Myxomatous degeneration of uterus, salpingitis	1	
Tubercular salpingitis, endometritis, peritonitis	1	
	—	14

Ovariectomy.

Papillomatous paroöphoritic cyst	2	
Dermoid cyst (retro-peritoneal)	1	
	—	3

Salpingo-oöphorectomy.

Chronic salpingitis and ovaritis	6	
Hystero epilepsy	1	
Tubo-ovarian abscess	2	
Tuberculosis of Fallopian tubes	1	
Pyosalpynx	1	
Hematosalpynx	1	
Hydrosalpynx	1	
	—	13
Ventrofixation of uterus for retrodisplacement with or without salpingo-oöphorectomy		11

Cœliotomy without Removal of Structures.

Tuberculosis of tube and ovary and general tuberculosis of peritoneum	1	
Cancer of fundus of a double uterus	1	
Epilepsy (suspected ovarian disease)	1	
Papillomatous cyst of ovary, general peritoneal papilloma	1	
Ventral hernia	1	
	—	5
Total		46

In the operations of ventrofixation of the uterus the primary object was the correction of a retrodisplacement. If in any case there were marked tubal and ovarian adhesions,

salpingo-oöphorectomy was performed. The operation of ventrofixation was always preceded by perineorrhaphy if there was present laceration or relaxation of the perineum. Drainage was employed six times in this series of cases for the following conditions:

Tubo-ovarian abscess—where streptococci, staphylococci, or bacillus coli commune were found in the pus.

Papillomatous cyst, with infection of the peritoneum.

Tuberculosis of the peritoneum.

Extra-uterine pregnancy.

NOTE ON STRONTIUM SALICYLATE.

By H. C. WOOD, M.D.

[Read December 5, 1894.]

I SHOULD like to say a word in regard to a drug which probably will prove a valuable addition to everyday therapeutics. After using the lactate, iodide, and bromide of strontium very freely, I came to the conclusion that the strontium element materially modifies the action of haloid bodies on the alimentary canal. This suggested the possibility that strontium might modify the action of salicylic acid, and I had Rosengarten & Sons, chemists, make strontium salicylate, and experimented with it upon dogs, determining that in therapeutic doses it elevates the arterial pressure, and that to depress the blood pressure and circulation larger amounts of it per kilo are required than of the sodium, or even of the ammonium salicylate. Subsequently to this study upon animals I have employed it largely in practice. I found, somewhat to my surprise, that in doses of 5 grains it is one of the best of intestinal antiseptics, yielding better results than salol, naphthalin, and similar agents. In doses of 10 or 15 grains it acts very decidedly as a salicylate in gouty and chronic rheumatic conditions without producing disturbance of the stomach. It may be given in capsules. I also found that when given largely it produces cinchonism, but it seems to be less active and powerful in acute cases than is the ammonium salicylate. In chronic gouty conditions and lithæmia, with intestinal indigestion, it appears to be the most valuable drug that we have.

A POMPEIAN CATHETER.

By JUDSON DALAND, M.D.

[Read December 5, 1894.]

The instrument shown is a *fac-simile* of a catheter which was found in a surgeon's chest, together with other surgical instruments, in the "House of Surgeons" in the ruins of Pompeii in 1819. The original has been deposited in the National Museum at Naples. It is tubular, made of bronze, measures ten and one-half inches in length, and has a double curve like an elongated letter "S," the distal end of which is closed and rounded. One-quarter of an inch from the distal extremity, on the superior aspect of this instrument, there is a lateral opening, ovoidal in shape, resembling very closely in position and shape the opening of the modern catheter. In size it corresponds to No. 18 of the French scale, and is very like a catheter often employed by Prof. Agnew. A female catheter of the same material, tubular, straight, and measuring three and one-half inches, was also found in the same case.

As the eruption of Vesuvius, which destroyed the city of Pompeii, occurred in the year 79 A.D., and as this catheter was found in an ordinary surgeon's chest in a town which at that time was of minor importance, it is plain that this instrument was in *general* use throughout that part of the world during and before that period. If the instrument had been invented in the year 79, its use would have been at first confined to the larger cities, whereas, as has been stated, it was found accidentally in Pompeii, a relatively small city, which then had a population of but 12,000.

The ancients employed bronze, copper, and silver in making this instrument, and later pewter, wood, and prepared leather were used.

No positive evidence exists as to the origin of the catheter, but numerous references are made to it in ancient Egyptian and Greek literature. Occasional references are made to the existence of a catheter in Chinese medicine many thousand years before the Christian era, but I have been unable to corroborate these statements. Prof. Morris Jastrow kindly looked through the Sanscrit, but nothing of importance could be discovered, and, with the kind assistance of Mrs. S. G. Stevenson, a similar search was made in Egyptian literature with the same results. I am at present in communication with the Curator of the British Museum, and hope to receive valuable information from that source.

There is evidence in literature that the catheter was first employed two thousand or three thousand years before Christ by the ancient Hindoos, who were at that time the most highly civilized nation in the world. In addition to other surgical instruments they employed catheters which were tubular and made in two shapes—first straight, and second with a single curve.

Hippocrates, in 460 B. C., states that it is an evidence of unskilfulness if a physician in catheterizing a patient does not succeed in entering the bladder.

The works of Celsus, Galen, and of authors in the sixth, tenth, and fourteenth centuries all prove that a knowledge of the catheter was never lost, despite J. Louis Petit's claim to have reinvented it in 1822.

REMARKS ON "*FAC-SIMILES* OF NINETEEN PERUVIAN SKULLS ILLUSTRATING ANCIENT TREPHINING."

By CHARLES W. DULLES, M.D.

[Made December 5, 1894.]

As stated by the Curator of the Mütter Museum, I wish to say a word to the College in regard to these skulls (or *fac-similes*) which has been suggested to me by a somewhat careful examination of them before the meeting. Of course, we must receive with respect the opinions of those who have already looked at these specimens, especially of the Surgeon-General of the Peruvian Army, and of the authorities of the Bureau of American Ethnology; but, none the less, it is proper for a body like the College to exercise its own judgment in drawing inferences from the facts which are before them. The difference between facts and inferences must always be borne in mind. About facts there should be no dispute; about inferences there may be much. The facts are that we have here *fac-similes* of certain skulls gathered, it is said, by Señor Manuel Antonio Muñiz, M.D, Surgeon-General of the Peruvian Army, who has selected them from a collection of about a thousand skulls, and that these *fac-similes* present certain marks and openings. The inference of Dr. Muñiz, and of others who agree with him, is that these marks and openings show that trephining was an operation practised in Peru in very early times—before the discovery of America by Columbus. The facts, this College may readily admit; but the inference may properly be questioned. I think that we may agree that

certain of the marks were probably made by human beings and during the life of the individuals from whom the skulls are derived. As to the instruments by which these marks were made, there may be a difference of opinion. It has been supposed that they were made by some sawing instrument used with a reciprocal motion. This instrument has been supposed to be a pointed bit of stone or arrow-head. The impression I get from these marks is, however, that they were made by some form of cutting instrument operated by a blow, such as the tomahawk, with an edge wedge-shaped in the perpendicular, and convex from end to end. In fact, to my eye, some of these marks are perfect moulds of sharp instruments of this sort, and utterly unlike the marks which would be produced by a sawing instrument, in which the width of the cut would be the same at both ends. A most important consideration in drawing inferences in regard to the skulls of which these are *fac-similes* depends upon the fact that they are casts made from moulds, and in this process we can understand that many details may have been lost, details which would give a distinct impression as to whether the instrument making these marks had operated so as to produce fracture or compression of the cancellated structure or of the vitreous table. As to the interesting specimens in which the marks are in pairs, the members of each pair being parallel and one pair crossing the other at right angles, it seems to me that it would be not unreasonable to suppose that these had been made in order to remove from the skull a weapon like a javelin, or an arrow, which was imbedded in it, and which could be freed by four such cuts. Others of the skulls look as though they might have been operated upon after death, in order to remove a piece of bone; for it is believed that ancient man used pieces of human bone as amulets or charms, or eyes for the images of his gods. In regard to the question as to the instruments used in making these sharp lines (which are exactly what would be made by a cutting edge like that of a tomahawk upon material calculated to take an exact impression of its edge) we may note that there is in one of these remarkable specimens what might

be called a superfluous mark, a little to one side of and about the same size as the others—a mark which would be inexplicable on the theory that the operator used an instrument so much under control of the hand as a sawing-instrument, but which is easy to explain on the supposition that, however skilful he may have been, one of the blows made with his tomahawk missed the mark and imprinted upon the skull a fortunate piece of proof in regard to what took place.

In regard to the inference that these skulls were operated upon surgically, and that the operation was that of trephining, it may be said that this term ought not to be applied in a different sense from that ordinarily employed. From the time of Hippocrates the trephine has been an instrument making a circular cut. What the ancients called trephining was an operation in which a round portion of bone was removed by means of a sort of perforator or terebrator, with a conical end notched like that of certain modern drills for metals, or by the modiolus, an instrument almost exactly like the saw of a trephine, both being set in motion by an appliance very much like that of the brace or auger of a carpenter at this day. On looking at the openings in these skulls we see that there is not one which bears any distinct evidence that it was made in this way. To assume that they were so made because some of them are irregularly rounded, is to assume too much. The inference that some of these openings were made ante mortem is reasonable, because the edges of the openings are such as we find to-day in the skulls of persons in whom Nature has attempted to repair disease or injury. But their appearance suggests to me the thought that such openings as these have been relied upon hitherto to prove that syphilis existed in America before the time of Columbus. Without committing ourselves to this inference, we may say that it is quite reasonable—indeed, more reasonable than the one with which these skulls come to us; for just such openings are made nowadays in the skull, and may be seen in museums as the consequence of syphilitic disease, and especially of erosion of bones by gummata. In this connection I exhibit some skulls taken from our Mütter Mu-

seum, showing on the one hand what kind of appearances are presented by modern trephine openings, and on the other the ravages of syphilis. This is not the time for elaborate discussion of this question; but I think the College may, with due respect to those who have regarded these specimens as illustrating ancient operations of trephining, conclude that this assumption is not clearly established by the facts before us. It is to be remembered, also, that among the relics found in Peruvian burial places there is none, I believe, resembling the trephine used by the ancients in Europe, or by modern surgeons, while the marks in certain of these skulls would accurately fit the edge of a stone hatchet.

It has been suggested that these cuts may have been produced by shells used in the manner of a saw; but this is only another hypothesis, and no hypothesis is strengthened by adding to it another one. How far this mode of reasoning may be carried is indicated in the present instance by the fact that one of these specimens is spoken of by W. J. McGee, of the Bureau of American Ethnology, as an example of trephining *for epilepsy*.

In view of the facts as they appear to me, I would suggest that the College accept these very interesting specimens without committing itself to the theory which comes to us with them.

REMARKS.

Dr. Guy Hinsdale: These *fac-similes* were made in Washington from the collection which was exhibited at the World's Fair by Señor Manuel Antonio Muñiz, Surgeon-General of the Peruvian Army. Among one thousand skulls obtained in different parts of Peru, these are the only ones that show evidences of these operations. Some of the openings evidently were produced by a sharp instrument before death, because an examination of the original specimens reveals evidences that healing has taken place. It is believed that they were done for surgical reasons, and also probably for the relief of some medical affections, as in one case, possibly, of epilepsy, and perhaps to relieve the individual from the influence of the evil spirit.

Several types of operation are shown. In one skull an extensive operation has been done, starting in front and passing to the back portion of the skull.

In this skull there was found a silver plate lying over the seat of operation. That is the only specimen ever known to have been covered by a silver plate.

Another specimen shows deep grooves, probably made by a stone or sharp piece of glass passed backward and forward until it was possible to remove portions of bone.

One specimen shows two large openings, one of which had evidently been survived for some years. It was a common practice among the Peruvians to remove buttons of bone from the skull, and these were then worn as charms. An individual dying, segments of the skull were cut out for this purpose. In one of these specimens an operation was evidently done on account of a fracture of considerable extent. The latter extends from near the centre of the occipital across the lower portion of the right parietal, and across the temporal, disappearing under the zygomatic arch. The original specimen, from a mummy, shows what is believed to be a scraped trephining yielding an aperture of about three-eighths of an inch in the occipital bone. The Museum is under obligations to Mr. Delancey W. Gill, of the Bureau of American Ethnology, for his efforts in securing an admirable reproduction of this interesting collection.

Dr. G. G. Davis: The main point of interest in this collection is the fact that it is a new contribution to our knowledge on this subject. In 1877 Broca wrote a very elaborate monograph on prehistoric trephining, and the main basis of his researches were skulls which had been found in Southern France. Later some skulls were found in Peru, but only one had an opening resembling that made by a trephine. This specimen was figured by Mr. Fletcher, of Washington, in a carefully-prepared study of the subject a few years ago. Since that time no contribution has come from South America until this collection was presented, and it forms a most important addition to the material on which our knowledge of the subject is founded.

With regard to the question of trephining by the laity, there is no doubt that they did trephine to a very elaborate extent, and that it is done even to the present day in the South Sea Islands. It is done on the living. The operation is performed in the native tribes of Algeria. Trephining was also done very frequently among the ancients.

When we come to the question whether the trephining in these specimens was done before or after death, I think that there is no doubt that the majority of these were done post mortem. The sharpness of the edges and marks of the tool used show that there was no attempt at repair. In the case of the skull covered with the plate the plate may have been put there to protect the brain, but it appears to me doubtful as to whether it was a living brain. One can see that there are a large number of scratches around the opening, and it shows that the process of repair had not been completed before the patient died; of course, he may have lived only a short time after the operation.

Here is one skull in which the evidences of healing are so marked that I have no doubt that it occurred during life; its edges are somewhat smoothed off, and the diploë is closed. In another the edges are sharp and the diploë still exposed, thus showing the patient did not live long, or that the operation was post mortem.

As Dr. Dulles has said, there is some question as to the mode in which these openings were made. In the mountains of Algeria the Kabyles, descendants of the Berbers, make a series of punctures with a brad-awl-like instrument, and then with a sharp tool run these holes into one, thus making a slit; by crossing these slits a piece is cut out of the bone. Mr. Fletcher has suggested that in some cases the opening was made with a triangular-like gouge similar to that used by engravers, called a burin. If these skulls are as ancient as they are supposed to be, it is hardly likely that such instruments were in use at the time the openings were made. I cannot believe that these openings were made by a blow directed from above downward against the bone, as has been suggested. It seems more reasonable to think that they were produced by a more or less circular or curved saw arrangement. Such an instrument could very well be made of flint or other hard substance. Very few of these specimens indicate that the process of scraping was resorted to. The South Sea Islanders particularly adopt that method of getting through the skull, and before the advent of the white man the sharp tooth of the shark was used. After his advent a piece of glass from a broken bottle was found to be a more efficacious and rapid implement, and was thereafter used for the purpose.

Dr. Abbott: As we are looking at copies, and not at the originals, it is impossible to reach a conclusion of any value as to the kind of instrument employed, and how it was used, or to say whether the operation was performed before or after death. In accepting these things, I think the College should see that they are conspicuously labeled as *casts only* of what has been assumed to be evidence of the early development stages of the operation of trephining. I should think it possible, by a careful examination of the originals, to say whether the openings in them were made sometime before or after death, and, perhaps, to say whether with an edged tool, saw, or similar instrument; but I do not consider one justified in expressing any opinion whatever upon these points from an examination of casts.

APPENDIX.

ADDITIONS TO THE MÜTTER MUSEUM.

Specimen illustrating Gastro-enterostomy. Presented by Dr. John B. Roberts.

Cancer of the Rectum. Presented by Dr. J. M. Baldy.

Pyosalpinx. Presented by Dr. J. M. Baldy.

Tubal Pregnancy. Presented by Dr. J. M. Baldy.

Primary Tuberculosis of the Spinal Arches. Presented by Dr. John B. Roberts.

Rudimentary Penis and Exstrophy of the Bladder. Presented by Dr. John B. Roberts.

Two Appendices. Presented by Dr. John B. Roberts.

Fatty Tumor from Bursa of the Knee. Presented by Dr. John B. Roberts.

Four Calculi. Presented by Dr. John B. Roberts.

Sac of Spina Bifida Treated by Excision. Presented by Dr. John B. Roberts.

Two Spinal Columns from Cases of Laminectomy. Presented by Dr. John B. Roberts.

Dermoid Cyst of the Ovary. Presented by Dr. John B. Roberts.

Tumor of the Bladder Removed by Suprapubic Lithotomy. Presented by Dr. John B. Roberts.

Pyosalpinx. Presented by Dr. John B. Roberts.

Cervical Tumor. Presented by Dr. John B. Roberts.

Enlarged Spleen from the Right Lumbar Region. Presented by Dr. John B. Roberts.

Two Gimlets used for fixing Excision of the Knee. Presented by Dr. John B. Roberts.

Specimens of Hysterectomy for Sarcoma. Presented by Dr. John B. Roberts.

Cancer and Dilated Stomach. Presented by Dr. John B. Roberts.

Lipoma of the Back. Presented by Dr. John B. Roberts.

Fibroid of the Ear. Presented by Dr. John B. Roberts.

Deformity of the Vomer. Presented by Dr. John B. Roberts.

Excision of the Thyroid Gland. Presented by Dr. John B. Roberts.

Lipoma. Presented by Dr. John B. Roberts.

Femoral Hernia. Presented by Dr. John B. Roberts.

Sarcoma of the Mamma. Presented by Dr. John B. Roberts.

Specimens from a Case of Exstrophy of the Bladder. Presented by Dr. John B. Roberts.

Double Hydrosalpinx. Presented by Dr. J. M. Baldy.

Ovarian Fibroid. Presented by Dr. J. M. Baldy.

Twenty Gunshot Fractures. Purchased.

Two Photographs of Hypertrophy of the Foot. By Dr. George E. Shoemaker.

Gumma of the Testicle. Presented by Dr. O. Horwitz.

Cystic Kidney. Presented by Dr. W. J. Taylor.

Fœtus. Presented by Dr. A. B. Hirsh.

Japanese Obstetric Hook. Presented by Dr. O. P. Rex.

Photograph of Prof. Politzer. Presented by Dr. C. H. Burnett.

Dermoid Cyst of the Ovaries and Fibroid of the Uterus. Presented by Dr. W. W. Ashhurst.

Three Photographs of the Gynecological Operating-room at Johns Hopkins Hospital. Presented by Dr. R. P. Harris.

Carcinoma of the Mamma. Presented by Dr. J. Ewing Mears.

Carcinoma of the Uterus. Presented by Dr. B. C. Hirst.

Ovarian Cyst. Presented by Dr. B. C. Hirst.

Hydrocephalic Fœtus. Presented by Dr. W. H. Morrison.

Cerebral Sarcoma. Presented by Dr. W. H. Morrison.

Specimens from Leprosy. Presented by Dr. J. C. Wilson.

Extra-Uterine Pregnancy. Presented by Dr. B. C. Hirst.

Album of Photographs. Presented by Mr. James F. Wood.

Mammary Tumor. Presented by Dr. H. A. Slocum.

Tubal Pregnancy. Presented by Dr. B. C. Hirst.

Dissection of Female Genitals to Show the Position of the Ureters. Presented by Dr. A. B. Hirsh.

False Aneurism of the Leg. Presented by Dr. H. R. Wharton.

Tubal Pregnancy. Presented by Dr. J. M. Baldy.

Multiple Fibroid of the Uterus. Presented by Dr. H. A. Slocum.

Baunscheidt's Instrument for Acupuncture. Presented by Dr. T. H. Andrews.

Supra-vaginal Elongation of the Cervix. Presented by Dr. J. M. Baldy.

Seven Specimens illustrating Intestinal Anastomosis; Experimental. Presented by Dr. J. M. Baldy.

Fac similes of Nineteen Peruvian Skulls, illustrating Aboriginal Trephining. Purchased.

Photograph of Sarcoma of the Neck. Presented by Dr. A. B. Hirsh.

Ovarian Cyst from a Patient aged Seventy. Presented by Dr. A. B. Hirsh.

Tubercular Kidney. Presented by Dr. Guy Hinsdale.

Male Pompeiian Catheter. Presented by Dr. Judson Daland.

Three Vesical Calculi. Presented by Dr. George M. Boyd.

ABSTRACT OF THE REPORT OF THE LIBRARY COMMITTEE.

The number of bound volumes now in the Library is 47,526, exclusive of duplicates and of the Hodge collection. Including these there is a total of 51,750 volumes. In addition to the bound volumes there are more than 26,000 unbound pamphlets, reports, and transactions.

The increase in the number of books during the year was 2515, which is greater by 629 than that of the preceding year. Of these accessions 608 were obtained by purchase, 151 by exchange, and the rest by gift.

The Library subscribes to 172 journals, and 430 current numbers are received by donation, making a total of 602.

Special attention has been given to completing the files of the more important journals and to obtaining theses and dissertations, and 377 volumes of journals and 796 French and German theses and dissertations have been purchased.

Seven hundred and eighty-one volumes have been bound during the year.

The number of visitors to the Library was 4575, an increase of 438 over that of the previous year. The number of books supplied by the Librarian for consultation was 8613, an increase of 1647; this is exclusive of the books taken from the shelves by the Fellows who have access to the cases. 2181 books were taken out, about the same number as during the previous year.

George C. Harlan,
Chairman.

LIST OF PAPERS: SECTION ON OPHTHALMOLOGY.

The following is the list of papers read and specimens, etc., exhibited at the meetings of the Section on Ophthalmology during the past year:

November, 1893.

Presentation of a Case of Blepharoplasty, by Dr. Harlan.

Exhibition of a Case of Plastic Operation for Ectropium, by Dr. Oliver.

Exhibition of Photograph showing Extensive Destruction of Cheek and Lid from Burn; Repaired by a Sliding Flap, by Dr. Norris.

Presentation of a Case in which Harlan's Operation and Skin Grafting had been done for Entropium and Symblepharon from Lime Burn, by Dr. Jackson.

Exhibition and Description of a Revolving Test Lens Frame, by Dr. Oliver.

Presentation of a Case of Double Iridectomy for Glaucoma, followed by Successful Extraction of an Opaque Lens, by Dr. Oliver.

Presentation of a Case of Double Neuro-retinitis, with Great Swelling of the Disk, by Dr. Thomas.

Presentation of a Case of Successful Magnet Extraction of a Piece of Steel from the Vitreous, by Dr. Jackson.

December, 1893.

Reports of Cases and Exhibitions of Diagrams of Fields of Vision:

I. Ring Scotoma in Glaucoma.

II. Monocular Ring Scotoma.

III. Central Scotoma, with Peripheral Contraction.

IV. Quadrant Defect passing into Ring Scotoma, by Dr. de Schweinitz.

Exhibition of a Triple Rotary Prism, by Dr. Jackson.

January, 1894.

Presentation of a Case of Albuminuric Retinitis, by Dr. Norris.

Presentation of a Case of Albuminuric Retinitis, by Dr. Jackson.

Exhibition of a Case of Areolar Choroiditis, by Dr. Oliver.

Exhibition of a Case of Successful Operation for Symblepharon, by Dr. Oliver.

Exhibition of a Case of Successful Operation for Ectropium, by Dr. Oliver.

Paper on the Practical Use and Value of the Ophthalmometer, by Dr. Jackson.

Presentation of an Improved Cell of Glass and Celluloid for the Preservation and Exhibition of Macroscopic Eye Specimens, by Dr. Oliver.

February, 1894.

Exhibition of Specimens and Photographs of Three Cases of Malignant Growth of the Orbit, by Dr. Harlan.

Exhibition of Specimens and Photographs of a Case of Melano-sarcoma of the Orbit, by Dr. Oliver.

Exhibition of Two Cases illustrating the Effects of Knapp's Roller Operation in Granular Conjunctivitis, by Dr. Oliver.

Presentation of a Case of Unusual Glaucomatous Cup, by Dr. Oliver.

Presentation of a Case of Traumatic Facial Paralysis, with Bilateral Abducens, Paralysis, Deafness, and Optic Nerve Degeneration, following Crush of the Head, by Dr. Oliver.

Presentation of a Case of Traumatic Cataract from the Blow of a Spent Bullet, by Dr. Oliver.

Exhibition and Description of a Set of Trial Frames for use with Prisms, by Dr. Risley.

March, 1894.

Presentation of a Case of Complete Closing of the Orbit after Removal of Contents for a Malignant Growth, by Dr. Shaffner.

Presentation of a Case of Nystagmus; Persistent Hyaloid Remains of a Double Coloboma of the Choroid, by Dr. Jackson.

Presentation of a Case of Multiple Traumatic Retinal Hemorrhage, by Dr. Oliver.

Paper on the Influence of Optical Defects on the General and Local Health, by Dr. S. Weir Mitchell.

April, 1894.

Exhibition of a Case of Double Retained Nerve Sheath, by Dr. Perkins.

Exhibition of a Case of Anterior Polar Opacity of the Lens, by Dr. Randall.

Paper on Anomalies of the Ocular Balance, by Dr. Risley.

Presentation of a Test for Ocular Balance at the ordinary distance for near work, by Dr. Jackson.

May, 1894.

Remarks upon Œdema and Cystoid Degeneration of the Retina, and Exhibition of Slides and Photo-micrographs, by Dr. Norris.

Exhibition of a Patient with Gumma of the Ciliary Body, by Dr. Harlan.

Exhibition of a Case of Recent Operation of Blepharoplasty, by Dr. Harlan.

Presentation of a Case of Traumatic Subluxation of the Lens and Rupture of the Choroid, by Dr. Jackson.

Description of a Case of Exophthalmic Goitre, by Dr. Oliver.

Presentation of a Case in which a Dislocated Lens producing Secondary Glaucoma had been successfully Extracted from the Anterior Chamber, with immediate cessation of all Pressure Symptoms, by Dr Oliver.

October, 1894.

Paper upon the Etiology and Treatment of Strabismus, by Dr. Hansell.

Description and Exhibition of Two Cases of Lipomatous Dermoid of the Eyeball, by Dr. de Schweinitz.

Exhibition and Description of a New Form of Optomyometer, by Dr. Oliver.

Exhibition of a Desk for the Use of Children with Progressive Near Sight, by Dr. Risley.

CHARLES A. OLIVER,
Clerk of the Section.

LIST OF PAPERS: SECTION OF OTOLOGY AND LARYNGOLOGY.

List of papers read and specimens presented in the Section of Otology and Laryngology of the College of Physicians from December 20, 1893, to December 11, 1894, inclusive:

December 20, 1893.

Dr. B. Alexander Randall exhibited a number of Casts of the Nose and its accessory cavities—the Pharynx, the Larynx and Bronchi, and the Auditory Canal, the Tympanic and Mastoid Cavities, and the Labyrinth.

Dr. J. Solis-Cohen, a paper on Sarcoma of the Tonsil removed through the Mouth; no signs of recurrence several months later.

February 6, 1894.

Dr. C. H. Burnett: A Case of Otitic Brain-abscess; operation by Dr. De Forest Willard; recovery.

Dr. B. Alexander Randall: A Case of Otitic Brain-abscess.

Dr. Harrison Allen: Rhinoliths.

March 6, 1894.

Dr. B. Alexander Randall exhibited a Patient in whom the Openings of the Sphenoidal Sinuses into the Rhino-pharynx could be distinctly seen with the Rhinoscope.

Dr. A. H. Cleveland (by invitation): Notes of a Case of Epithelioma of the Uvula.

Dr. Alexander Watt MacCoy: Mouth-breathing.

Dr. Charles Shaffner: Ruptures of the Membrana Tympani.

Dr. Charles H. Burnett: Three Cases of Chronic Tinnitus Aurium and Tympanic Vertigo relieved by removal of the Incus.

April 3, 1894.

Dr. B. Alexander Randall exhibited a Patient with Ambilateral Suppuration in the Attic and Perforation of the Membrana Flaccida.

Dr. B. Alexander Randall: The Over-use of Instrumental Aids to Exploration in Otology.

Dr. E. Baldwin Gleason (by invitation): Exhibition of a Case of Supposed Ankylosis of the Malleus to the Incus, in a Woman.

Dr. L. J. Hammond (by invitation): Three Cases of Excision of the Membrana Tympani for Attic Suppuration, followed by Facial Paralysis.

May 1, 1894.

Dr. B. Alexander Randall exhibited a Case of Pseudo-membranous Rhinitis, without faucial lesions, in which cultures seemed to prove the presence of the Klebs-Loeffler bacillus.

Also, a Case of Bilateral Acute Attic Suppuration as bearing upon the symmetrical occurrence of the Foramen Rivini.

Dr. E. B. Gleason (by invitation) exhibited anatomical preparations, viz.: Anomalous Membranous Septum in the Antrum and Ankylosis of the Stapes.

Dr. Harrison Allen: Verbal Communication as to Sex in Relation to Disease of the Nose, citing conditions found in only one sex or the other, as constituting a little-studied field.

Dr. C. H. Burnett: Persistent Mobility of the Stapes in some cases of Chronic Catarrhal Deafness.

October 2, 1894.

Dr. C. H. Burnett: Chronic Purulent Otitis Media, with Facial Paralysis; Sequestra from the Auditory Canal.

Dr. A. H. Cleveland (by invitation): Notes of a Case of Chronic Otitis Media Purulenta Dextra; extensive Sinus Thrombosis of *both sides*.

Dr. Ralph W. Seiss: The Remote Results of Intra-nasal Operations.

November 6, 1894.

Dr. E. L. Vansant: A Case of Syphilitic Cicatricial Adhesions of the Tongue to the Palate and Pharyngeal Walls, with notes of operations.

Dr. E. L. Vansant exhibited a Patient with a largely Cleft Palate very successfully closed with a Hard-rubber Obturator.

Dr. B. Alexander Randall exhibited a Case of Epithelioma of the Ear of thirty years' duration, with photographs of conditions five and seven years previously.

Dr. Alexander Watt MacCoy: Observations on certain pathological changes in the Middle Turbinal Structures.

Dr. Harrison Allen exhibited a New Nasal Electrode of the smallest possible size, and arranged to work by mere contact with the handle without clamping.

Dr. B. Alexander Randall: The Anatomical and Pathological Relations of Stacke's Operation, with demonstration of normal and morbid specimens.

December 11, 1894.

Discussion upon the best form of Illumination for Examinations of the Ear and Throat at the bedside.

Dr. J. Solis-Cohen: A Case of Successful Removal of Fibro-sarcoma of the Rhino-pharynx, with specimen.

Dr. A. H. Cleveland (by invitation): Carcinoma of the Antrum of Highmore, with specimens.

Dr. J. S. Gibb: Report of Two Cases of Septal Abscess.

CHARLES H. BURNETT,
Clerk of the Section.

LIST OF PAPERS: ORTHOPEDIC SECTION.

October 20, 1893.

Pathology of the Paralysis of Caries of the Spine. Charles W. Burr, M.D.

Pachymeningitis from Pott's Disease, and other Secondary Infective Processes, especially Neuritis of the Cauda Equina from Bedsore over the Sacrum. Exhibition of Segment of the Spine and Microscopic Section of the Spinal Cord from an Obscure Case. J. Hendrie Lloyd, M.D.

Pathology and Treatment of Paralysis due to Pott's Disease. Wharton Sinkler, M.D.

The Surgical Treatment of Caries of the Spine. W. W. Keen, M.D.

The Mechanical Treatment of Paralysis of Caries of the Spine. William J. Taylor, M.D.

November 17, 1893.

Hallux Valgus: Exhibition of Patient showing the Results of Operation. John B. Deaver, M.D.

Casts taken from a Severe Case of Hallux Valgus. H. Augustus Wilson, M.D.

Rheumatic Ankylosis of Ankles, Knees, Hips, Elbows, and One Wrist. Exhibition of Patient. Thomas G. Morton, M.D.

Report of a Case of Suppuration of the Knee-joint and Evacuation of Pus; Recovery, with Useful Joint. W. Joseph Hearn, M.D.

December 15, 1893.

Spasmodic Torticollis and its Medical Relations. F. X. Dercum, M.D.

Report of Two Cases of Spasmodic Torticollis Cured by the Administration of Conium. Wharton Sinkler, M.D.

Exhibition of Patient with Torticollis following Caries of the Cervical Vertebræ. H. Augustus Wilson, M.D.

January 19, 1894.

A Case of Primary Tuberculosis of the Laminæ and Spinous Processes of the Vertebral Column. John B. Roberts, M.D.

The Use of a Brace in the Treatment of Lateral Curvature resulting from Empyema. G. G. Davis, M.D.

A Case of Deformity of the Hip Corrected by Subcutaneous Osteotomy. Henry R. Wharton, M.D.

Report of Two Cases of Congenital Hypertrophy. T. S. K. Morton, M.D.

Congenital Hypertrophy and the Report of a Case of Hypertrophy of the Foot. George Erety Shoemaker, M.D.

February 16, 1894.

Sarcomatous and Other Tumors which may in the Early Stages be Confounded with Hip Disease. De Forest Willard, M.D.

Report of a Case of Concurrent Sarcoma and Hip Disease. J. Torrence Rugh, M.D. (by invitation).

Exhibition of a Case in which Gangrene of the Toes Occurred in a Case of Caries of the Hip and Lumbar Vertebræ. H. Augustus Wilson, M.D.

March 16, 1894.

A Spinal Case for Diagnosis. Presented by F. X. Dercum.

Apparatus for Preventing and Correcting Deformity after Excision of the Knee. G. G. Davis, M.D.

Report of a Case of Right-angle Flexion and Rotation following Excision of Tuberculous Knee. H. Augustus Wilson, M.D.

November 16, 1894.

The Treatment of Rotary Lateral Curvature of the Spine. Illustrated by photographs, specimens, etc. E. H. Bradford, M.D., of Boston (by invitation).

Wm. J. Taylor,
Clerk of the Section.

LIST OF PAPERS: SECTION IN GENERAL SURGERY.

The Section in General Surgery was organized March 9, 1894, and held five meetings during the remainder of the College year, at which papers were read as follows:

March 9, 1894.

Report of Operation for Ancient Dislocation of the Elbow, by Dr. John B. Roberts.

Resection of Elbow for Old Dislocation, by Dr. George E. Shoemaker.

Delayed Union in Fractures, by Dr. Thomas G. Morton.

Multiple Excisions of Inferior Maxillary Nerve, by Dr. L. W. Steinbach.

Syphilitic Orchitis, by Dr. Orville Horwitz.

Some Recent Experiences with Tumors of the Liver, by Dr. John B. Roberts.

Enormous Oval Hemorrhoid Encircling the Anus: Whitehead Operation, by Dr. W. W. Keen.

April 13, 1894.

Misplaced Testicle Restored in its Proper Position, by Dr. Edward Martin.

Neuritis of Long Respiratory Nerve of Bell, by Dr. Edward Martin.

Comminuted Fracture of Atlas, with Rupture of Vertebral Artery and almost entire absence of Primary Symptoms, by Dr. Thomas S. K. Morton.

Demonstration of the Workings of a New Surgical Engine, by Dr. M. H. Cryer.

The Marriage of Syphilitics, by Dr. William G. Porter.

May 11, 1894.

Fracture of the Humerus during Kocher's Manipulation for Old Dislocation, by Dr. Thomas S. K. Morton.

Fracture of Humerus under Kocher's Method of Reduction, by Dr. L. W. Steinbach.

New Surgical Ward Carriage, by Dr. Thomas G. Morton.

Two Cases of Appendicitis Complicated by Glycosuria, by Dr. Joseph Leidy.

A Case of Appendicitis Complicated by Glycosuria, by Dr. Thomas S. K. Morton.

Observations upon Appendicitis, by Dr. J. William White. Discussion opened by Associate Fellow Robert F. Weir, of New York.

October 12, 1894.

Improved Lithotrites, and Demonstration of Measured Strength of Lithotrites and Crushing Resistance of Calculi, by Dr. William F. Forbes.

Four Cases of Abdominal Section for Traumatism, by Dr. Robert G. LeConte.

Case of Gunshot Wound of Abdomen and Thorax, by Dr. L. W. Steinbach.

Case of Gunshot Wound of Liver and Lung, by Dr. Thomas S. K. Morton.

November 9, 1894.

Wood-pulp Splints and Splint-making Materials, by Dr. William J. Taylor.

Two Cases of Thyroideetomy, by Dr. John B. Roberts.

Report of Cases of Diphtheria Treated by the Antitoxine, by Dr. George Muehleck.

THOMAS S. K. MORTON,
Clerk of the Section.

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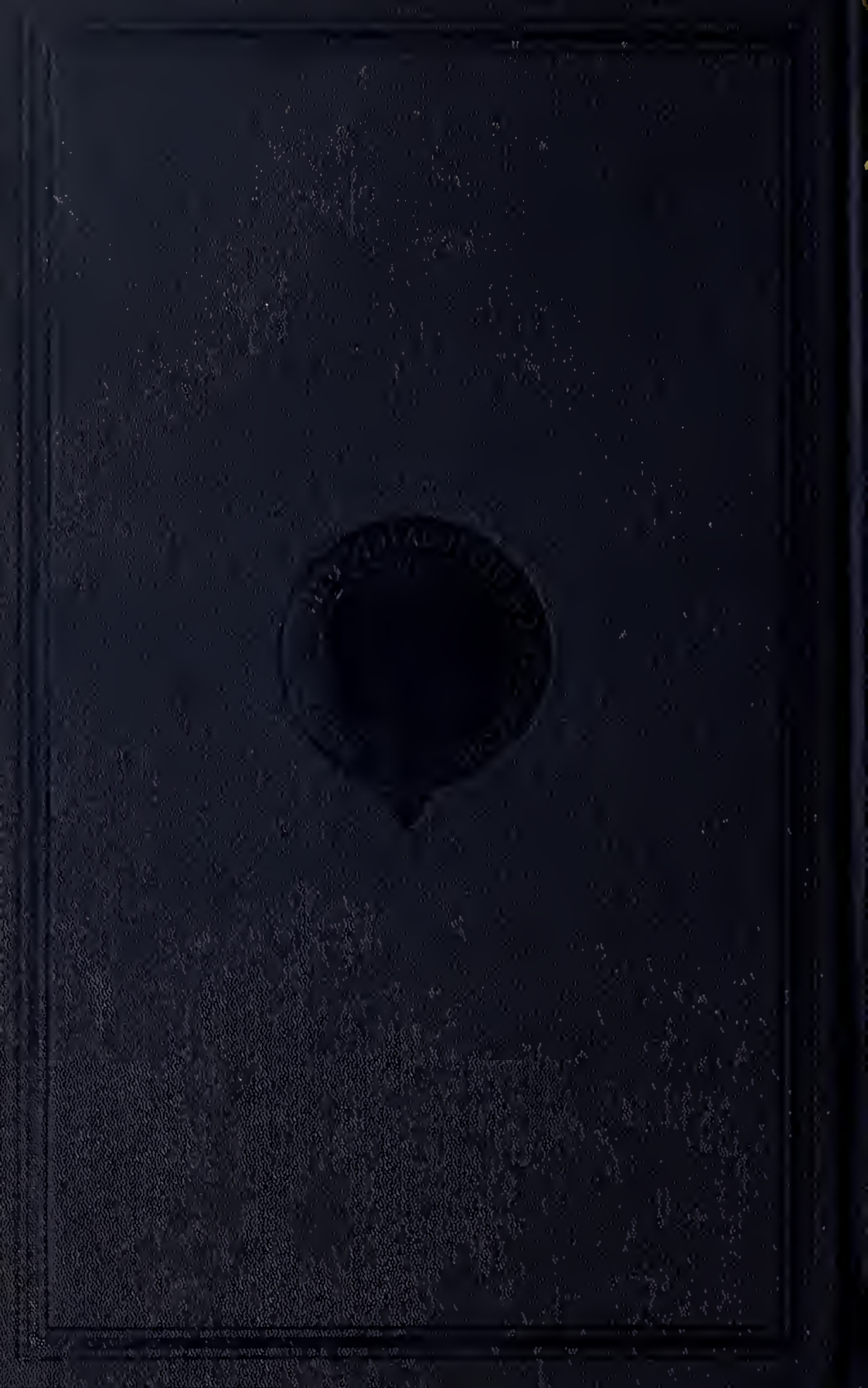
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